A STUDY TO ASSESS THE EFFECT OF TREADMILL ON INTRAOCULAR PRESSURE IN MEDICAL STUDENTS IN THE AGE GROUP OF 18-25 YEARS

DISSERTATION SUBMITTED

BY

DR. T.M.SATHISHKUMAAR

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY

IN

OPHTHALMOLOGY

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

APRIL 2015

DEPARTMENT OF OPHTHALMOLOGY

PSG INSTITUTE OF MEDICAL SCIENCES AND RESEARCH

COIMBATORE

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I hereby declare that this dissertation entitled **"A STUDY TO ASSESS THE EFFECT OF TREADMILL ON INTRAOCULAR PRESSURE IN MEDICAL STDENTS IN THE AGE GROUP OF 18-25 YEARS"** is a bonafide and genuine research carried out by me under the guidance of **DR.D.SUNDAR, D.O.M.S** Professor, Department of Ophthalmology, PSG Institute Of Medical Sciences And Research, Coimbatore in partial for the award of M.S Degree in Ophthalmology to be held in 2015. This dissertation has not been submitted in part or full to any other university or toward any other degree before this below mentioned date.

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Place: Coimbatore **Dr.D.SUNDAR M.S.D.O,**

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ACKNOWLEDGEMENT

This thesis would never have been made possible without the valuable help and constant support of my professors, colleagues and family members.

Like a street lamp lighting the pathway at night my respected guide and Head of Department **Dr. SUNDAR,** Department of Ophthalmology, PSG Institutes of Medical Sciences and Research, has guided me along the path to completion of this dissertation and my three year life in PSG.

The contribution of **Dr. RADHAKRISHNAN** Professor, Department of ophthalmology, PSG Institutes of Medical Sciences and Research, who has subsequently left our institution, has been a pillar as long as he was here and even after he has left. His absence is highly felt and regretted by all of us in the department.

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A silent and watchful teacher and a friend **Dr. DIVYA**, Assistant Professor, Department of Ophthalmology, PSG Institutes of Medical Sciences and Research, has helped me to perfect my thesis and clinical skills while contributing to my attitude and outlook on patients.

I would also like to thank the new assistant professors in our beloved department **Dr. LEKHA** and **Dr. ALO SEN**.

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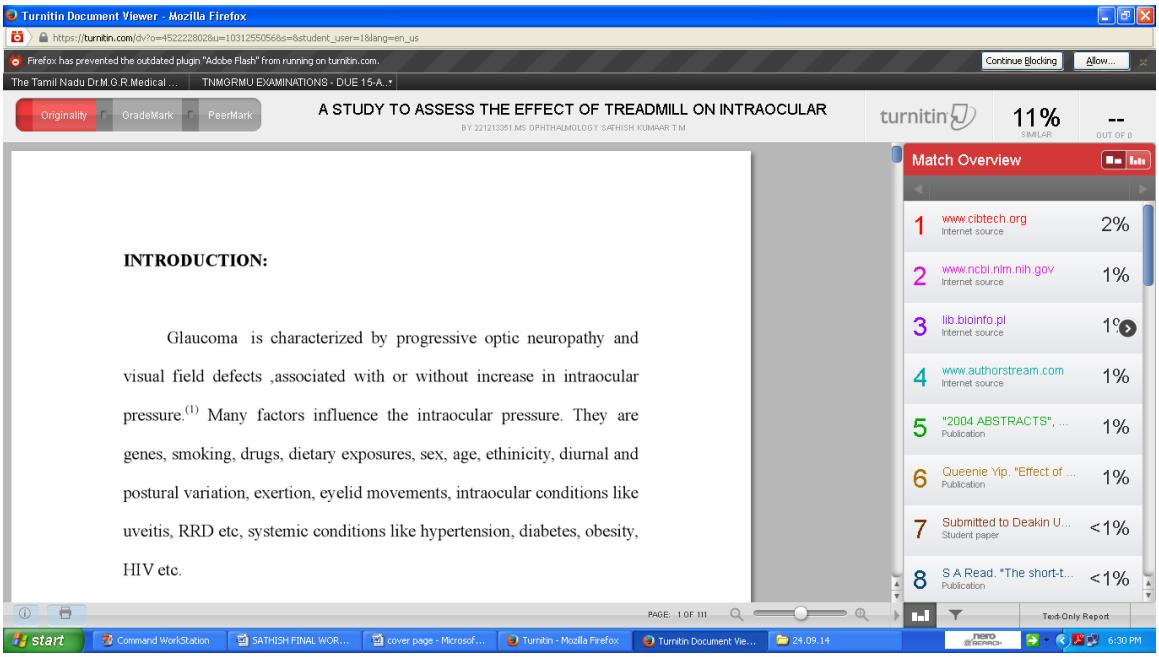
The omniscient and omnipresent heavenly God, has watched over this thesis and me for all my life. Thank you Almighty.

I hope that everyone I have encountered in my life in this institution would always be present till the last of my days as friends and guides.

I thank you all.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INTRODUCTION:
Glaucoma is characterized by progressive optic neuropathy and visual field defects ,associated with or without increase in intraocular pressure.(1) Many factors influence the intraocular pressure. They are genes, smoking, drugs, dietary exposures, sex, age, ethinicity, diurnal and postural variation, exertion, eyelid movements, intraocular conditions like uveitis, RRD etc, systemic conditions like hypertension, diabetes, obesity, HIV etc.

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October 22, 2013

To
Dr T M Sathishkumaar
Postgraduate
Dept. of Ophthalmology
PSG IMS & R
Coimbatore

Ref.: Proposal titled: *"A study to assess the effect of treadmill on intra ocular pressure in medical students in the age group of 18 - 25 years"*

Sub.: Ethics Committee Approval for the study

The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on 1st October, 2013 in its full board review meeting held at College Council Room, PSG IMS&R, between 2.00 pm and 4.30 pm, and discussed your application to conduct the study entitled:

"A study to assess the effect of treadmill on intra ocular pressure in medical students in the age group of 18 - 25 years"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Informed Consent forms in English and Tamil
4. Permission letter from the Head of the Institution
5. Data Collection Tool
6. Budget
7. CV

The members who attended the meeting at which your study proposal was discussed are as follows:

Sl. No.	Name of the Member of IHEC	Qualification	Area of Expertise	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
1	Dr. S. Bhuvaneshwari (Member-Secretary, IHEC)	MD	Clinical Pharmacology	Female	Yes	Yes
2	Mrs. Geetha S Kannan	MA	Lay person	Female	No	Yes
3	Mr Gowpathy Velappan	BA., BL	Legal Advisor	Male	No	Yes
4	Mrs G Malarvizhi	M Sc	Nursing	Female	Yes	Yes
5	Mr. R. Nandakumar (Vice-Chairperson, IHEC)	BA., BL	Legal Expert	Male	No	Yes
6	Dr. G. Rajendiran	DM	Clinician (Cardiology)	Male	Yes	No
7	Dr. V. Ramamurthy	Ph D	Biotechnology	Male	Yes	No
8	Dr. M. Ramanathan	M Pharm, Ph D	Non-Medical (Pharmacy)	Male	Yes	No

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9	Dr. P. Sathyan (Chairperson, IHEC)	DO, DNB	Clinician (Opthalmology)	Male	No	Yes
10	Dr. Seetha Panicker	MD	Clinician (Obstetrics & Gyanaecology)	Female	Yes	Yes
11	Dr. S. Shanthakumari	MD	Pathology, Ethicist	Female	Yes	No
12	Dr. Y.S. Sivan	Ph D	Social Scientist (Sociology)	Male	Yes	Yes
13	Dr. Sudha Ramalingam (Alternate Member-Secretary, IHEC)	MD	Public Health, Epidemiology, Genetics, Ethicist	Female	Yes	Yes
14	Mrs. K. Uma Maheswari	M Sc, M Phil. B Ed	Botany	Female	No	Yes
15	Dr. D. Vijaya	M Sc, Ph D	Basic Medical Sciences (Biochemistry)	Female	Yes	Yes

After due consideration, the committee has decided to approve the above proposal.

The approval is valid for one year.

We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

We hereby confirm that neither you nor any of your study team members have participated in the voting/ decision making procedure of the committee. The members of the committee who have participated in the voting/ decision making procedure of the committee do not have any conflict of interest in the referenced study.

This Ethics Committee is organized and operates according to Good Clinical Practice and Schedule Y requirements.

Non-adherence to the Standard Operating Procedures (SOP) of the Institutional Human Ethics Committee (IHEC) and national and international ethical guidelines shall result in withdrawal of approval (suspension or termination of the study). SOP will be revised from time to time and revisions are applicable prospectively to ongoing studies approved prior to such revisions.

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Request for renewal must be made at least a month ahead of the expiry of validity along with a copy of the progress report.

23-10-17

Dr S Bhuvaneshwari
Member - Secretary
Institutional Human Ethics Committee

SECRETARY
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INSTITUTIONAL HUMAN ETHICS COMMITTEE

TABLE OF CONTENTS

INTRODUCTION :

Glaucoma is characterized by progressive optic neuropathy and visual field defects ,associated with or without increase in intraocular pressure.[1] Many factors influence the intraocular pressure. They are genes, smoking, drugs, dietary exposures, sex, age, ethinicity, diurnal and postural variation, exertion, eyelid movements, intraocular conditions like uveitis, RRD etc, systemic conditions like hypertension, diabetes, obesity, HIV etc.

HISTORY OF GLAUCOMA

Glaucosis refers to the bluish-green hue of the affected eye.[2] Hippocrates quotes glaucoma as blindness coming in advanced years and is associated with a glazed appearance of the pupil. In the Arabic era , At-Tabari (tenth century) in the "Book of Hippocratic treatment" explained that glaucoma is a chronic inflammatory condition of the eye with raised intra ocular pressure. Sams-ad-Din (1348) of Cairo described among the one hundred and fifty three diseases of the eye and its adnexa, ophthalmias as a migraine of the eye or "headache of the pupil an illness associated with pain in the eye hemicrania and dullness of the humours, and followed by dilatation of the pupil and cataract, if it became chronic, tenseness of the eye and blindness supervened[2]. The revolution took

place in Nineteenth century when Antoinne-Pierre Demours (1818) suggested glaucoma as a raised ocular tension . The clinical picture was fully detailed and he described for the first time the appearance of the colours of a rainbow around the lights. In London Guthrie GI described glaucoma as hardness of the eye. Ophthalmoscope Made a new revolution in the history of glaucoma, when clinical observation on the glaucomatous cup began to accumulate, the disease was divided into three categories

1) Acute

2) Chronic and

3) Secondary by von Graefe (1857) with the help of ophthalmoscope.

Formation of peripheral anterior synechiae was placed higher up in the ladder as the main source of obstruction of aqueous humour drainage resulting in primary open angle glaucoma for many decades by many ophthalmologists.

With the advent of the gonioscope, various others pointed out that glaucoma could be divided into two types

1) Open angle glaucoma

2) Closed angle glaucoma

SIGNIFICANCE:

Glaucoma, the world wide leader for irreversible blindness and the successor of cataract for bilateral blindness. World Health Organization in 1995 published the data which reveals that glaucoma was prevalent among 5.1 million people around the world and accounting for 13.5% of global blindness. Open angle glaucoma and angle closure glaucoma was expected to be prevalent in 66.8 million people accounting for 6.7 million bilateral blindness by the year 2000 as described by the study done in 1996. According to the recent data, By the year 2010 Around 60.5 million persons will be affected world wide by glaucoma. By the year 2020 it is estimated that 79.6 million people will be affected by glaucoma world wide. [3]

ETHNIC DIFFERENCE:

S.NO	PARAMETERS	AMERICANS	INDIANS
1	Prevalence 2010	3.3 millions	11.9 millions

ANATOMICAL CONSIDERATIONS

Aqueous humour dynamics revolves around intraocular pressure maintenance and glaucoma pathophysiology. Ciliary body, posterior chamber, anterior chamber, angle of the anterior chamber and the aqueous outflow system are the principal ocular structures concerned with aqueous humour dynamics. Aqueous humour which is produced in the posterior chamber by Pars plicata of the ciliary body (which is forward continuation of the choroid at the ora serrata), flows through the pupil to the anterior chamber and ultimately drains in to the Peripheral recess or angle of the anterior chamber[4]

I. ANGLE OF ANTERIOR CHAMBER:

Clinically the Gonioscopic examination reveals the following angle structures from posterior to anterior:

1. THE CILIARY BAND

The anterior most part of the ciliary body between its attachment to the scleral spur and insertion of iris forms the ciliary band , which acts as the posterior most structure in the angle recess.

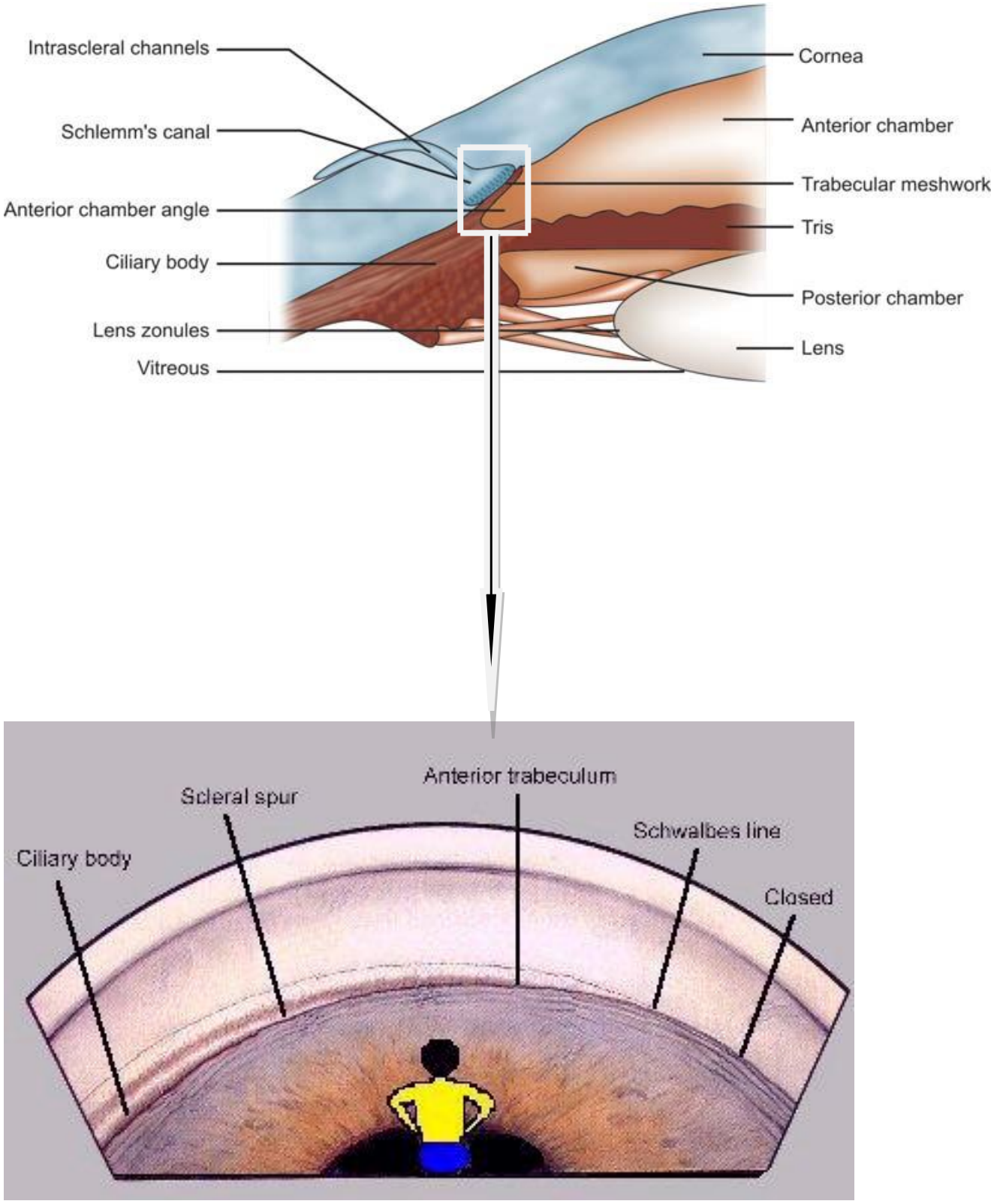


Fig:1: shows the angle of anterior chamber

2. SCLERAL SPUR

The sclera spur is formed by the scleral roll [group of fibres] runs parallel to the limbus and projects inwards. 75 to 80% collagen and 5% elastic tissue constitutes the scleral spur roll.[5]

3. TRABECULAR MESHWORK

Trabecular meshwork converts the scleral sulcus into a circular channel called as schlemm's canal. It consists of

a) Uveal meshwork
b) Corneoscleral meshwork
c) Juxtacanalicular tissue (cribriform layer).[6]

UVEAL MESHWORK:

From the iris root and ciliary body to the peripheral cornea there extends bands or rope like trabeculae referred to as uveal meshwork which consists of irregular openings sizing up from 25 to 75 µm across.

CORNEOSCLERAL MESHWORK:

From the scleral spur to the lateral wall of the scleral sulcus extends the corneoscleral meshwork which consists of flat sheets of trabeculae perforated by elliptical openings of sizes ranging from 5 to 50 microns.

JUXTACANALICULAR TISSUE:

It is the most important part of trabecular meshwork which offers the normal resistance to the outflow and represents the outermost part of trabecular meshwork.[7]

4. SCHWALBE'S LINE

In front of the trabecular meshwork, the prominent end of the Descemet's membrane of the cornea forms a ridge called as Schwalbe's line which marks the anterior limit of the structures forming the angle of the anterior chamber.[8]

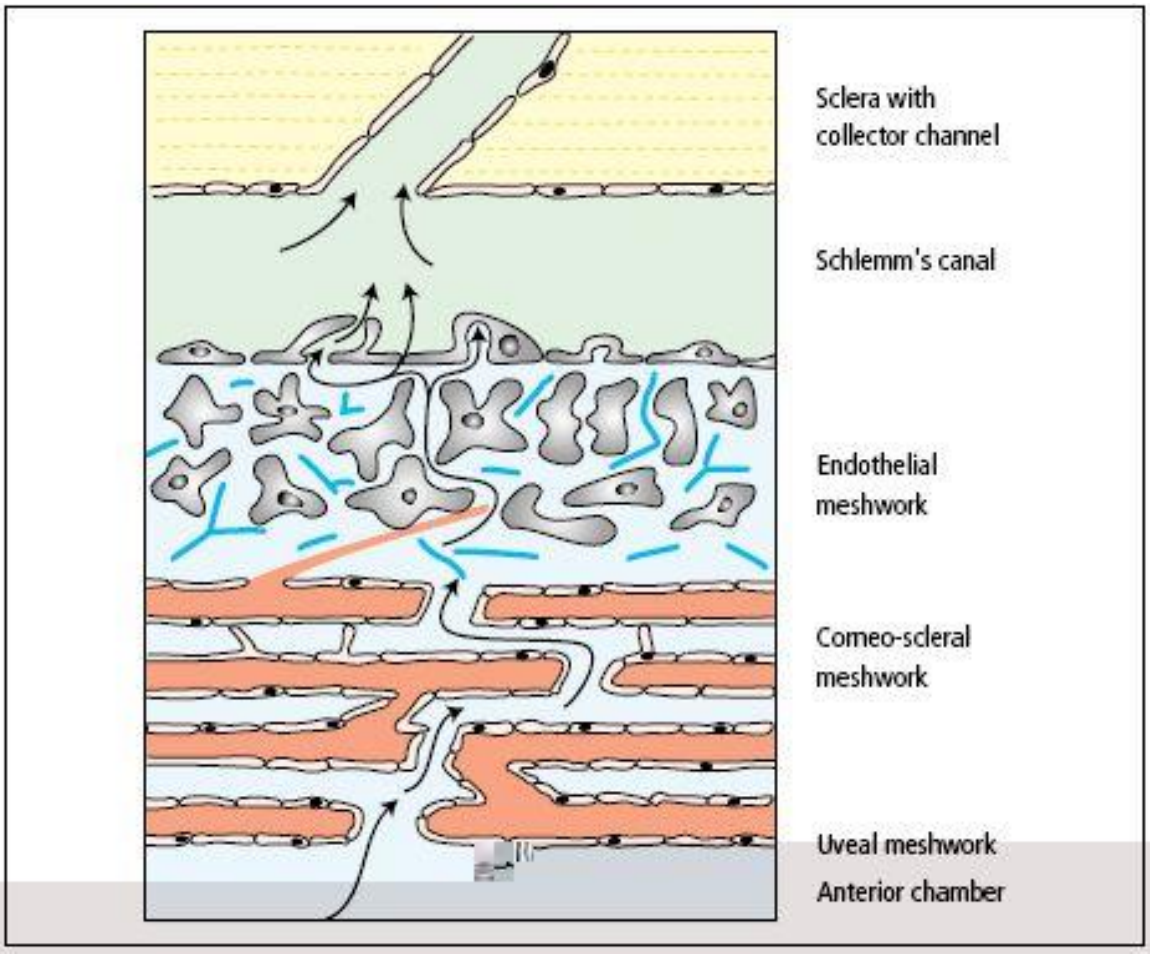


Fig:2: shows the parts of trabecular meshwork.

AQUEOUS HUMOUR:

Aqueous humour dynamics revolves around intraocular pressure maintenance and glaucoma pathophysiology. From posterior chamber of the eye and through out the anterior chamber of the eye there circulates the clear colourless watery solution called as aqueous humour. A dynamic equilibrium exists with regarding to aqueous humour helps to maintain its continuous circulation.

I. PHYSIOLOGICAL CONSIDERATIONS:

1. FORMATION:

a) Diffusion

b) Ultra filtration

c) Secretion

FORMATION OF AQUEOUS HUMOUR:

The plasma within the capillary network of ciliary process acts as the source of aqueous humour.[9] For reaching the posterior chamber the aqueous humour travels across the capillary wall, the stroma and the two layers of epithelium by the following processes:[10]

DIFFUSION:

Diffusion is a process in which there is movement of the particles from areas of higher concentration to areas of lower concentration to maintain the uniform concentration in the space in which they are contained. Here in the production of aqueous humour the lipid substances travels across the cell membrane by a potential concenteration gradient. [11]

DIFFUSION:

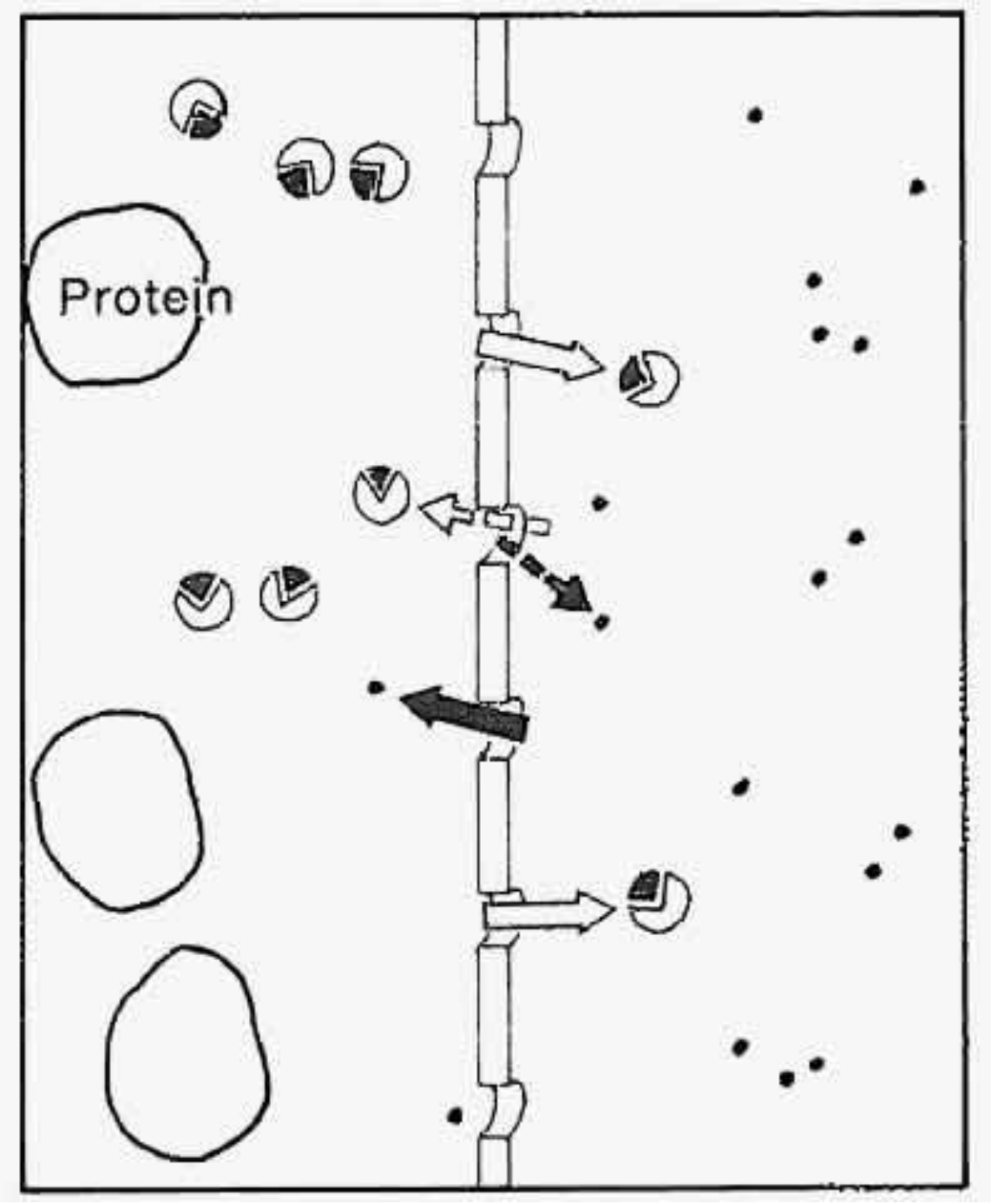


Fig:3: shows the process of diffusion.

ULTRAFILTRATION:

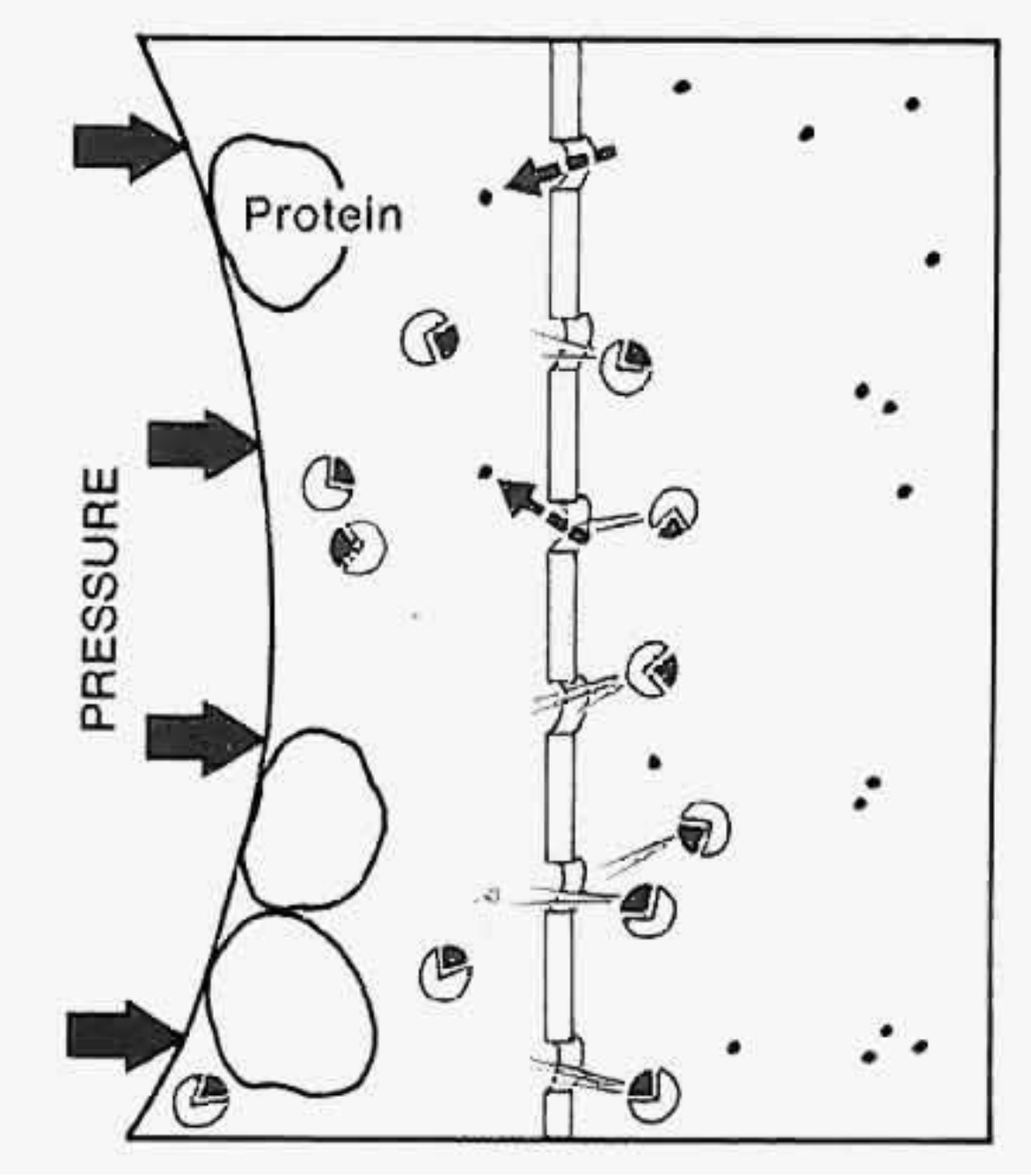


Fig:4: shows the process of ultrafiltration

Dialysis is the process in which there is separation of protein and salt from plain water by a membrane permeable to salt and water and not to protein by diffusion. Ultrafiltration is a process of dialysis under hydrostatic pressure. Here in the process of aqueous formation the size and charge determined water soluble substances and water flows with the help of osmotic gradient or hydrostatic pressure across the micropores in the protein part of the cell membrane.[12]

SECRETION:

Secretion is an active process which is unique because as the rest of processes are passive in nature. It is active because there is movement of particles from areas of lower concentration to areas of higher concentration which is against the concentration gradient with the expenditure of energy. Here in the process of aqueous formation larger size or highly charged water soluble substances are actively transported across the cell membrane the globular proteins in the cell membrane are responsible for this mechanism.[13] During this process there is expenditure of huge amount of energy.

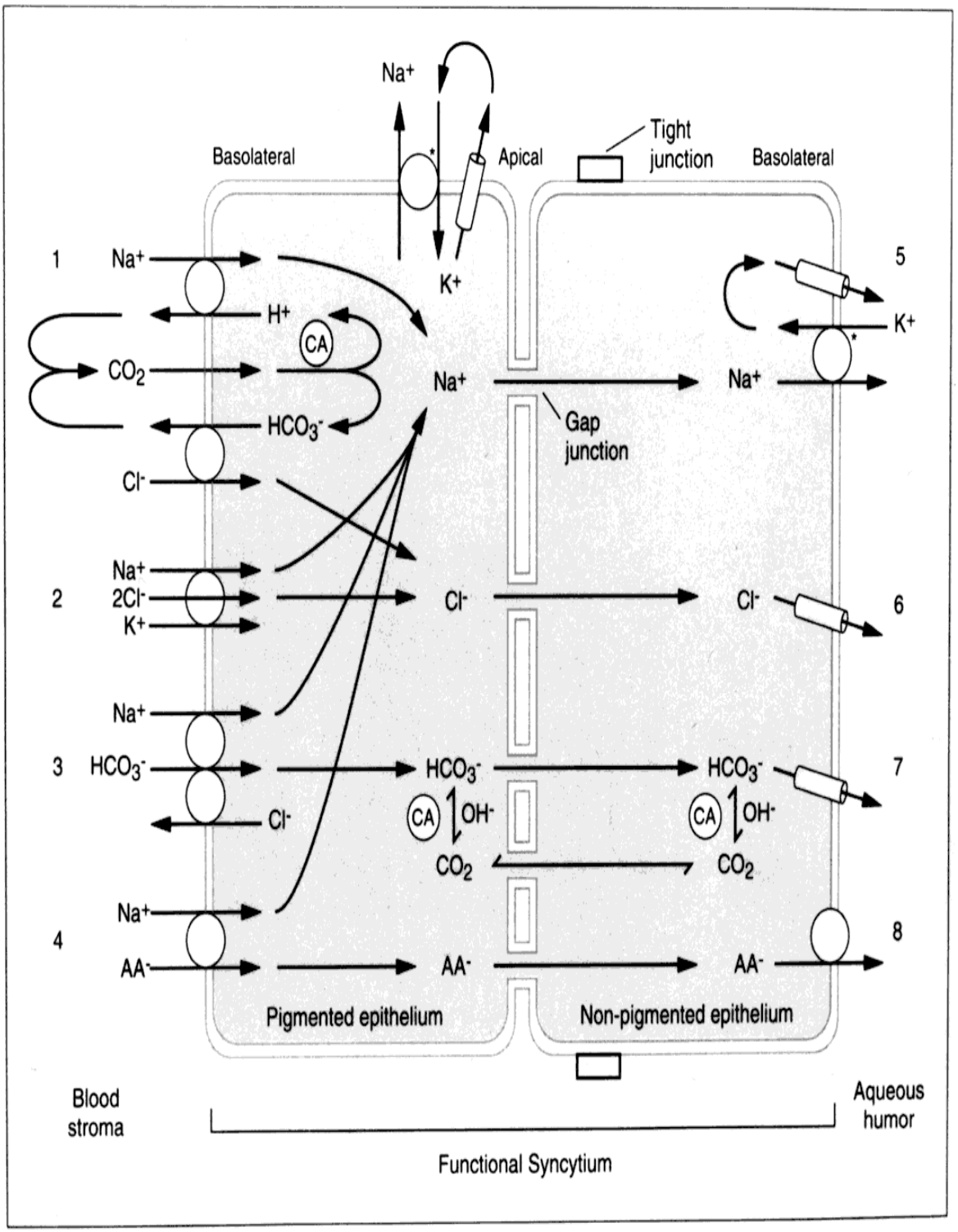


Fig:5: shows the process of secretion

2. FUNCTIONS:

a) Maintenance of intraocular pressure is the most important function of aqueous humour. By this the shape and internal structure of eye is maintained.

b) METABOLIC FUNCTION:

- Addition of substrates and removal of metabolites from the avascular ocular structures is done by aqueous. In cornea glucose and oxygen are taken up from aqueous, carbondioxide and lactic acid are released in to aqueous.
- Oxygen, glucose , aminoacids and potassium are taken up by lens from aqueous, lactate , pyruvate and sodium are released in to aqueous.
- Vitreous and retinal metabolism: Amino acids and glucose are passed into the vitreous from the aqueous

c) OPTICAL FUNCTIONS:

- Normally aqueous is optically clear. The diverging lens of low power is maintained by the corneal aqueous interface.

d) Blood, blood cells, lens matter, macrophages, products of inflammation are all cleared by the aqueous humour from the eye.

II. PHYSICAL AND CHEMICAL PROPERTIES:

1) The volume of the aqueous humour in the anterior chamber is about 0.25 ml and the volume of the aqueous humour in the posterior chamber is about 0.06 ml. so in toto the volume of aqueous humour in the eye is about 0.31 ml.
2) Refractive index of aqueous humour is 1.336. this RI is lower than that of cornea , so as a result the light rays slightly diverge when they pass through the corneo aqueous interface.
3) Aqueous humour density is less than that of water. The viscosity is about 1.025 to 1.040 with relative to water.
4) the osmotic pressure is 3 – 5 mosm/lit higher than that of plasma.
5) The pH is acidic in nature which is about 7.2.
6) The aqueous is produced at the rate of 2.3 microlit/min.

III. BIOCHEMICAL COMPOSITION:

Regarding aqueous humour formation, the old concept is based on leber's theory which states that simple filtration from the blood is responsible for aqueous humour formation but the biochemical

composition of aqueous humour reveals that the aqueous humour formation is multifactorial and cannot be explained by a single theory.

IV. BIOCHEMICAL CONSTITUENTS:

1) Water: 99% of the aqueous humour is constituted by water.
2) Proteins: protein content differentiates the aqueous humour from the plasma. In plasma 6-7 grams/100ml is present whereas in aqueous humour 5 -16 mg of proteins/100 ml is present.
3) IgG and IgM immunoglobulins are present in human aqueous. Plasminogen and its proactivators are present. FGF, TGF- beta and IGF are also present. The protein content difference between the plasma and aqueous is seen because of the presence of blood aqueous barrier.
4) Aminoacids: the free aminoacids ranges with the plasma from 0.08 – 3.14.
5) Non colloidal constituents: absolute clear nature of aqueous humour is due to the presence of dissolved solids which are called as non colloids table
6) Inulin and steroids are present in aqueous humour which enters the aqueous humour by diffusion.
7) Prostaglandins are actively secreted in to the aqueous from the iris.

8) Cyclic AMP level in the aqueous humour is equal to that of the plasma.

1. FACTORS AFFECTING THE COMPOSITION OF AQUEOUS HUMOUR:

A) Blood ocular barrier

B) Hemodynamic factors

C) Diffusion

D) Metabolites

E) Rate of aqueous drainage

F) Neurohormonal factors

A) BLOOD OCULAR BARRIER:

For effective vision of the eye, clarity in the media of the eye is essential. To maintain this clarity the blood ocular barrier plays a significant role which in turn alters the composition between the blood compartment and ocular compartment. The blood ocular barrier prevents the diffusion of large molecular sized components from entering the ocular cavity.

The blood ocular barrier is divided in to 2 parts. They are

i) blood retinal barrier

ii) blood aqueous barrier

i) BLOOD RETINAL BARRIER:

It is divided in to 2 parts. They are

- Inner blood retinal barrier
- Outer blood retinal barrier

- **INNER BLOOD RETINAL BARRIER:**

Tight junctions of retinal capillaries and endothelial cells forms the inner blood retinal barrier.

- **OUTER BLOOD RETINAL BARRIER:**

Zonula occludens and zonula adherans located between the retinal pigment epithelium are responsible for outer blood retinal barrier.

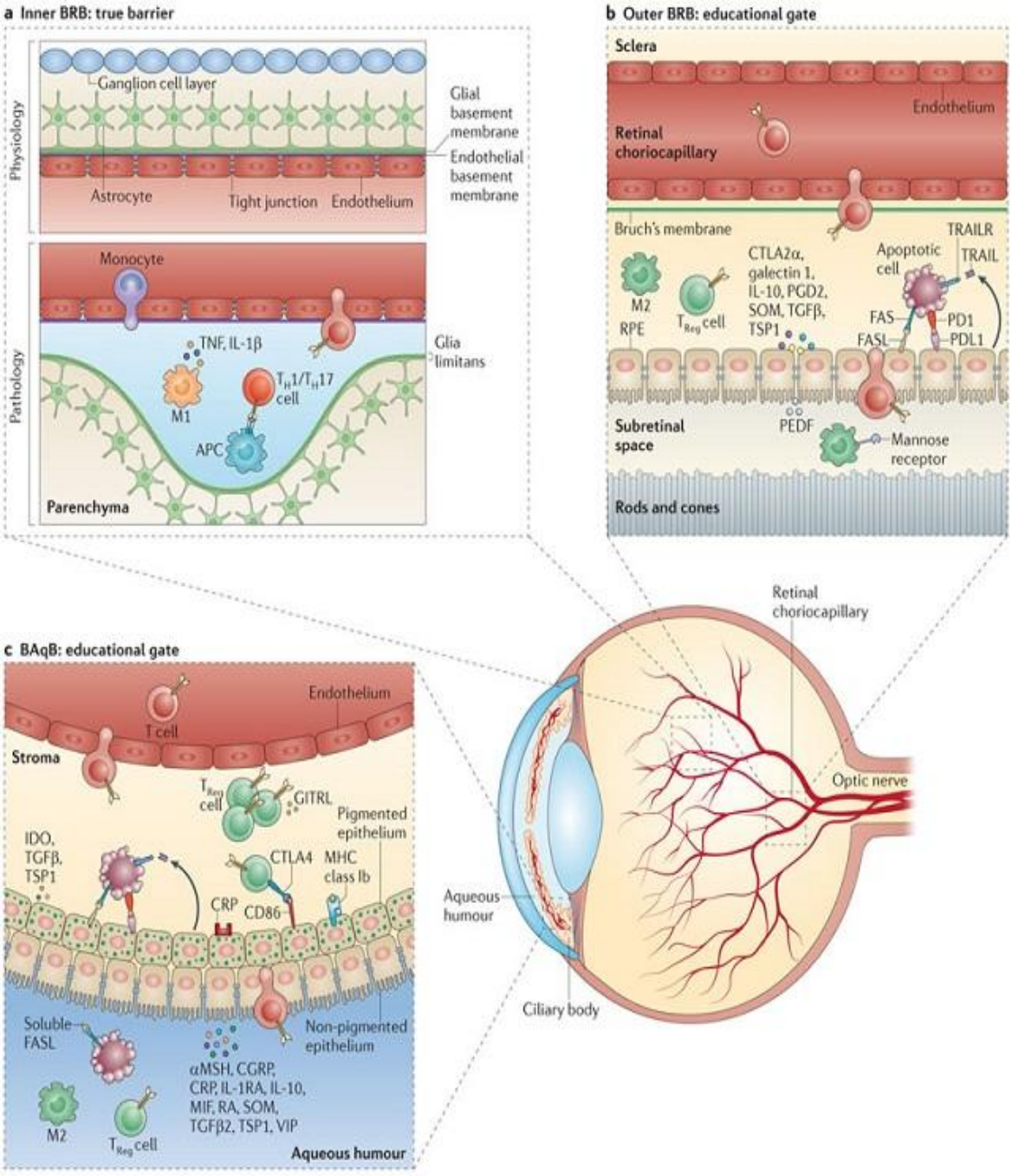

a Inner BRB: true barrier
Physiology
Ganglion cell layer
Glial basement membrane
Endothelial basement membrane
Astrocyte
Tight junction
Endothelium
Pathology
Monocyte
TNF, IL-1β
Glia limitans
M1
T_H1/T_H17 cell
APC
Parenchyma
b Outer BRB: educational gate
Sclera
Endothelium
Retinal choriocapillary
Bruch's membrane
TRAILR
Apoptotic cell
TRAIL
M2
RPE
T_{Reg} cell
CTLA2α, galectin 1, IL-10, PGD2, SOM, TGFβ, TSP1
FAS
FASL
PD1
PDL1
Subretinal space
PEDF
Mannose receptor
Rods and cones
c BAqB: educational gate
Endothelium
T cell
Stroma
T_{Reg} cell
GITRL
Pigmented epithelium
IDO, TGFβ, TSP1
CTLA4
MHC class Ib
CRP
CD86
Soluble FASL
Non-pigmented epithelium
αMSH, CGRP, CRP, IL-1RA, IL-10, MIF, RA, SOM, TGFβ2, TSP1, VIP
M2
T_{Reg} cell
Aqueous humour
Retinal choriocapillary
Optic nerve
Aqueous humour
Ciliary body

Fig:6: shows the inner and outer blood retinal barrier

ii) BLOOD AQUEOUS BARRIER:

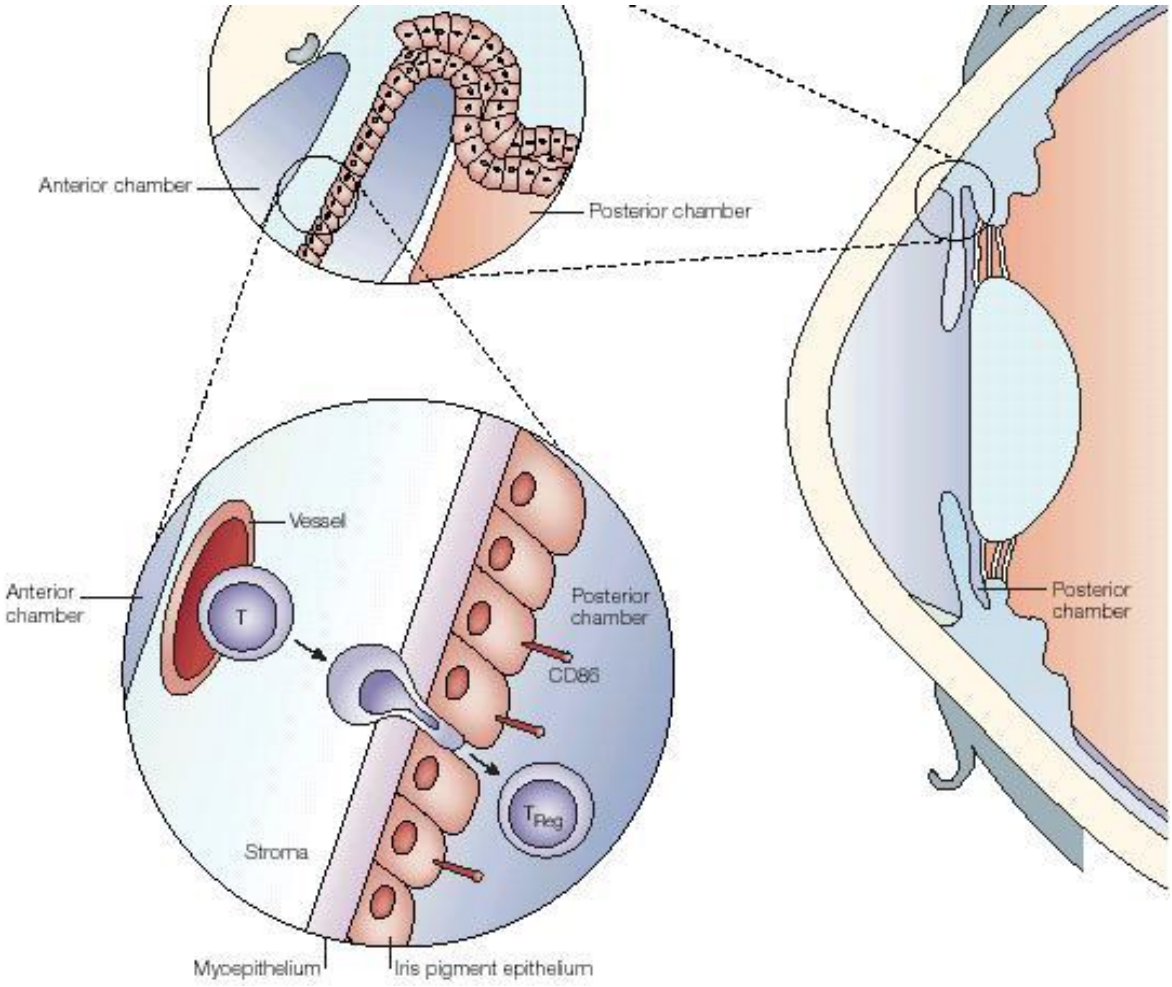


Fig:7: shows the blood aqueous barrier

It is formed by the tight junctions between the non fenestrated endothelium of iris capillaries and inner non pigmented epithelium of ciliary body. In inflammatory conditions the endothelial cells of iris becomes leaky.

Blood ocular barrier is not complete and absolute. Medium sized and water soluble substances transit across the capillary wall at a slower rate in to the ocular compartment. The lipid soluble nature also facilitates the lipid soluble substances to penetrate in to the aqueous.

V. APPLIED ASPECT OF BLOOD OCULAR BARRIER:

1) After the Supplementation of mannitol, mannitol poorly crosses the blood ocular barrier but its composition rises in the extracellular compartment thereby dehydrating the ocular compartments. So ultimately there is a fall in intraocular pressure due to the hyperosmolar nature of mannitol.

2) Ampicillin, chloramphenicol and cephalothin are the low molecular weight antibiotics which can cross the blood ocular barrier.

VI. BLOOD AQUEOUS BARRIER BREAKDOWN:

FACTORS:

a) OCULAR TRAUMA:

i) Mechanical

- Paracentesis
- Corneal abrasion
- Intraocular surgery
- Stroking iris

ii) Physical

- x-rays
- atomic radiation

iii) Chemical

- Alkali
- Irritants, eg. Nitrogen mustard

b) PATHOPHYSIOLOGIC

i) Vascular dilatation

- Histamine

- Sympathectomy

ii) Corneal and intraocular infections

iii) Intraocular inflammations

iv) Prostaglandins

v) Anterior segment ischemia

c) PHARMACOLOGIC

i) Melano stimulating hormone

ii) Nitrogen mustard

iii) Cholinesterase inhibitors

iv) Cholinergic drugs

v) Plasma hyperosmolality

2. EFFECT:

a) Secondary aqueous formation

b) Increase in protein content which is viewed through the slit lamp microscope as a pronounced tyndall beam.

c) The ionic composition equates with plasma

d) Fibrinogen appears in aqueous which causes clotting of aqueous

e) Diagnostic marker to find out the integrity of blood ocular barrier: The rapid entry of Evans blue dye and flourescein can be used as a diagnostic marker for the breach in the blood ocular barrier.

B) HEMODYNAMIC FACTORS:

The hemodynamic factors influencing the ciliary process stroma (stromal pool) because ultrafiltration which is the second step in aqueous humour production takes place in the ciliary process stroma.

C) DIFFUSION:

The iris vessels are easily permeable to electrolytes and anions so the diffusional exchange across the iris plays a significant role in the aqueous humour composition.

D) METABOLITES:

The metabolic process taking place in the cornea, retina , lens and vitreous will also alter the aqueous humour composition.

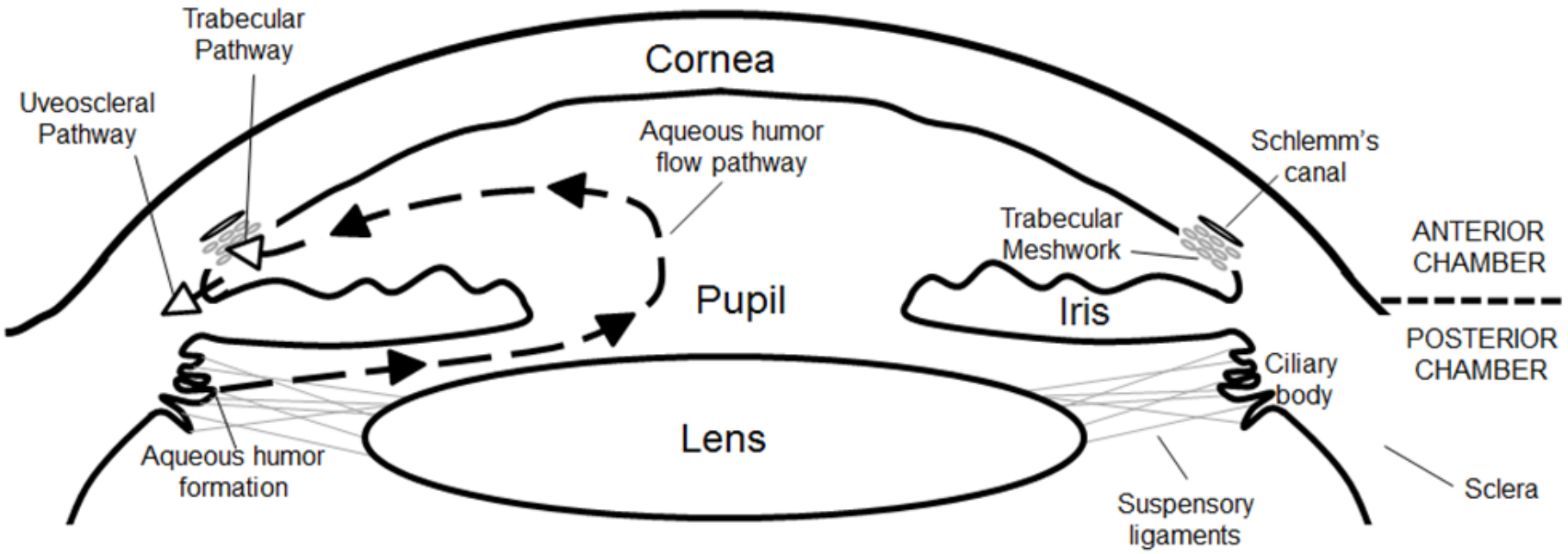
E) RATE OF AQUEOUS HUMOUR DRAINAGE:

Since a dynamic equilibrium exists between production and drainage of aqueous humour increase and decrease in rate aqueous humour drainage will greatly influence the composition of aqueous humour.

F) NEUROHORMONAL FACTORS:

Neurohormonal factors altering the aqueous humour formation process and its influencing factors , will also alter the quality of aqueous humour which inturn will have a effect on aqueous humour composition.[(4)]

I) AQUEOUS HUMOUR DYNAMICS:



Flg:8: shows the aqueous outflow pathways

The anterior chamber is seen in two aspects as the anterior and posterior part. The anterior part is cooler than the posterior part because the posterior part is warmed up by the vascular activity of the iris and the anterior part is cooled down by the avascular nature of the cornea and the evaporation of tears. This temperature gradient which causes the thermal current which in turn regulates the aqueous humour outflow.

There are 2 types of outflow uveoscleral outflow and trabecular outflow. They are

1) Uveoscleral outflow
2) Trabecular outflow

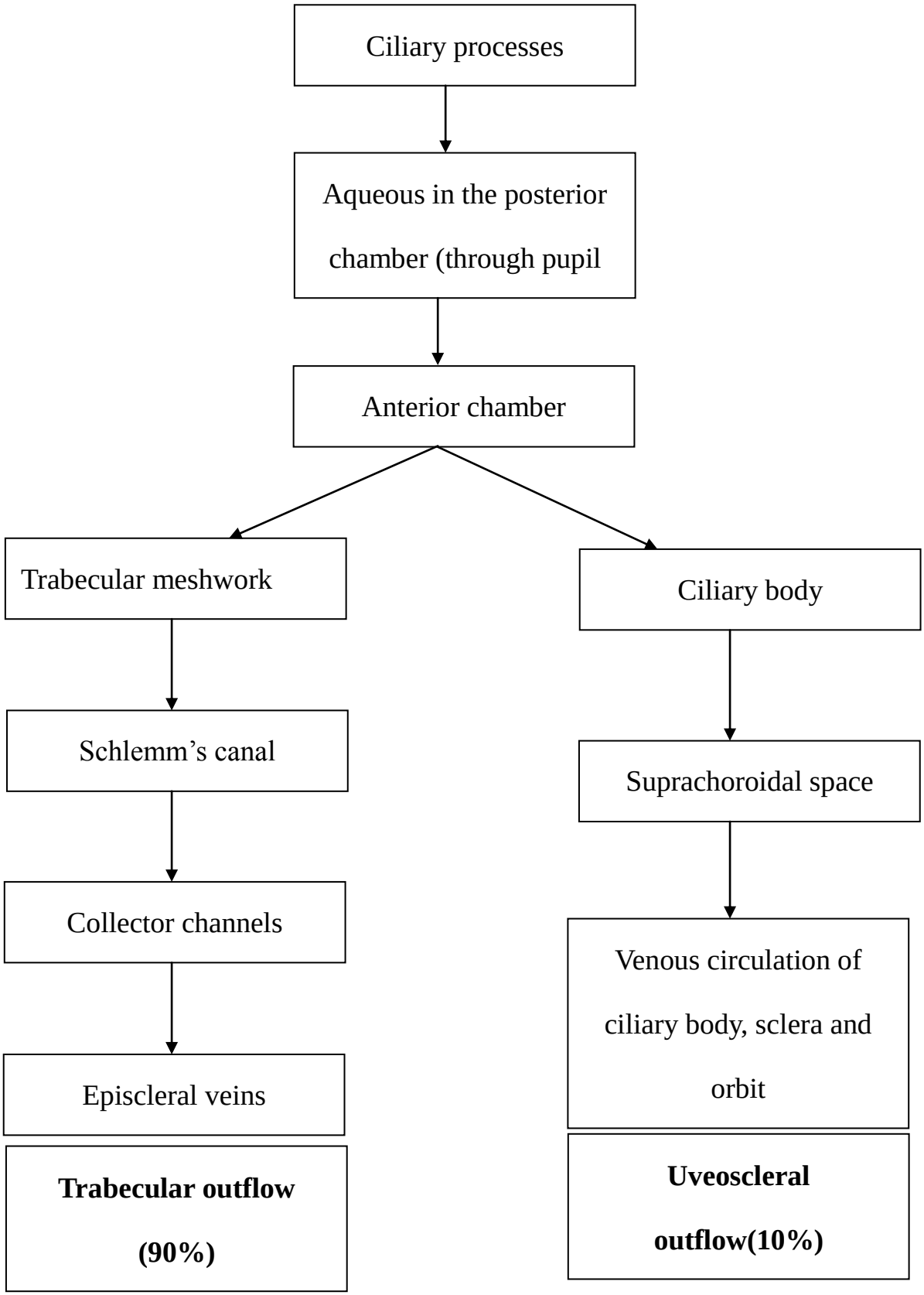
UVEOSCLERAL OUTFLOW:

It is responsible for 10-25% of total aqueous outflow. It drains approximately 0.3 microlit/minute which is independent of intraocular pressure.

TRABECULAR OUTFLOW:

75-90 % of the total aqueous outflow is drained via this route which forms main outlet of aqueous humour. Aqueous freely flows till juxtacanalicular meshwork which appears to provide mild resistance to the outflow.[(4)]

FLOWCHART DEPICTING SUMMARY OF AQUEOUS HUMOUR DRAINAGE



II) VARIOUS MECHANISMS OF AQUEOUS TRANSPORT:

1) Vacuolation theory [pores and giant vacuole system]
2) Leaky endothelial cells
3) Sonderman’s channels
4) Contractile microfilaments
5) Pores in endothelial cells

III) AQUEOUS OUTFLOW SYSTEM

The trabecular meshwork, Schlemm's canal, collector channels, aqueous veins and the episcleral veins forms the aqueous outflow system.[14]

1) TRABECULAR MESHWORK:

Trabecular meshwork converts the scleral sulcus into a circular channel called as schlemm’s canal. It consists of

1) Uveal meshwork
2) Corneoscleral meshwork
3) Juxtacanalicular tissue (cribriform layer).

2) SCHLEMM'S CANAL:

In the sclera sulcus ,a circumferential endothelial lined oval channel is present which is called as schlermm's canal.[15]The outer wall of the canal contains the numerous openings of the collector channels.

3) COLLECTOR CHANNELS:

25 to 35 intrascleral aqueous vessels are present at oblique angles which are called as collector channels to terminate in episcleral veins.

4) EPISCLERAL VEINS:

The above mentioned Collector channels terminate in to episcleral veins which drains in to anterior ciliary and superior ophthalmic veins. These veins ultimately drains in to cavernous sinus.

IV) INTRAOCULAR PRESSURE:

Normal intra ocular pressure may be defined as the pressure which does not lead to glaucomatous damage of the optic nerve head. The normal IOP ranges between 10 to 21 mm of hg. The mean IOP is 15.8 ±2.57 mm of Hg[16] and the two standard deviation above the mean was above the 20.5 mm of Hg under which 95% of the area approximately

lies under the Gaussian curve. IOP is a complex trait which is determined also by aqueous humour flow, uveoscleral outflow, trabecular outflow and episcleral venous pressure[10-15 mm of Hg].[17-22]

FACTORS AFFECTING IOP:

As mentioned earlier, IOP is a complex trait so we are dividing the factors affecting IOP under three categories

1) Genetics

2) Environment

3) Physiology

1) GENETICS:

IOP is a quantitative trait.[23,24] Monozygotic twins studies regarding IOP had more correlation than dizygotic twins studies. Twin twin pair studies shows more correlation than twin spouse studies.[25,26] IOP has strong hereditary inheritance,[27-34] so evaluating the gene loci a "major gene" was quoted in blue mountain eye study[35], 10q22 was showed in a family study[36], 5q22 and 14q22[37] were accounted in affected sibling pair study, the Beever dam eye study showed the involvement of 2,5,6,7,12,15 and 19 chromosomes were being linked to IOP.[38] However the recent studies enumerate that there is a mixture of genes influence on IOP thereby creating a major and minor influence on

IOP variation and variation in the glaucoma medication response to IOP.

2) ENVIRONMENT:

Four environmental factors are documented here.

A) Physical

B) Smoking

C) Drugs

D) Diet

PHYSICAL :

Episcleral venous pressure is decreased on exposure to cold air which subsequently leads to decrease in intraocular pressure.[39] IOP is increased when the gravity is decreased because of the shift in intravascular and extravascular body fluids towards the cephalad direction. [40]

SMOKING:

IOP is increased immediately after smoking because of the vasoconstriction taking place in the episcleral veins which leads to increase in episcleral venous pressure subsequently leading to increase in IOP.[41]

C) DRUGS:

DRUGS WHICH DECREASES IOP:

1) General anaesthetics except ketamine[42,43]
2) Heroin
3) Marijuana

DRUGS WHICH INCREASES IOP:

1) Succinyl choline
2) Suxamethonium
3) LSD[44,45]
4) Corticosteroids
5) Anticholinergic agents
6) Sulphonamide drugs

DRUGS WHICH PRONE TO CAUSE ANGLE CLOSURE GLAUCOMA:[46]

1) Scopolamine dermal patches
2) Aerosolized atropine
3) Ipratropium

Evaluation of infants ,children, patients with ocular trauma and patients with rupture globe is to be done with serious consideration to the fact of anaesthesia induced alteration in intraocular pressure. Congenital glaucoma patients evaluated under general anaesthesia is the main concern to avoid the artificial reduction of intraocular pressure which could mask the pathological rise in intraocular pressure. The extrusion of ocular contents due to sudden elevation of intraocular pressure is the primary concern when operating on an injured eye due to peneteration or during intraocular surgeries.[47]

D) DIET:

Acute doses of alcohol lowers intraocular pressure.[48-50] Caffeine causes slight rise in intraocular pressure.[51] Fruits and vegetables causes 69% decreased risk of glaucoma in reference to womens health study comprising 1155 participants.[52] Omega 3 fatty acids decreases intraocular pressure.[53]

3) PHYSIOLOGY:

The mean intraocular pressure in the older age groups is seemed to be rised with their increase in age in women who attained menopause

than men. The increase in standard deviation of intraocular pressure distribution is equal in men and women of white population. Japanese study revealed no sex difference.[54] intraocular pressure was higher among women than men in Barbados eye study.[55] The sex difference has no significant influence on intraocular pressure in the age group between 20 to 40 years of age. intraocular pressure increases with increase in age.

4) ETINICITY:

COAG is more common among blacks and asian population has more prevalence of angle closure glaucoma.[56-58]

5) DIURNAL AND POSTURAL VARIATION:

Intraocular pressure shows dynamic diurnal and postural variation. The diurnal variation of intraocular pressure is regulated by ACTH hormones and catecholamines but not by the influence of melatonin. The diurnal variation of intraocular pressure is substantiated with the study conducted on 1062 middle aged and older patients showing higher intraocular pressure during day time,[59] a study on 690 diurnal curves of intraocular pressure showed 40% of patients with elevated intraocular pressure in the early morning and 65% of patients showed high intraocular pressure before noon. [60]

6) EXERTIONAL INFLUENCE:

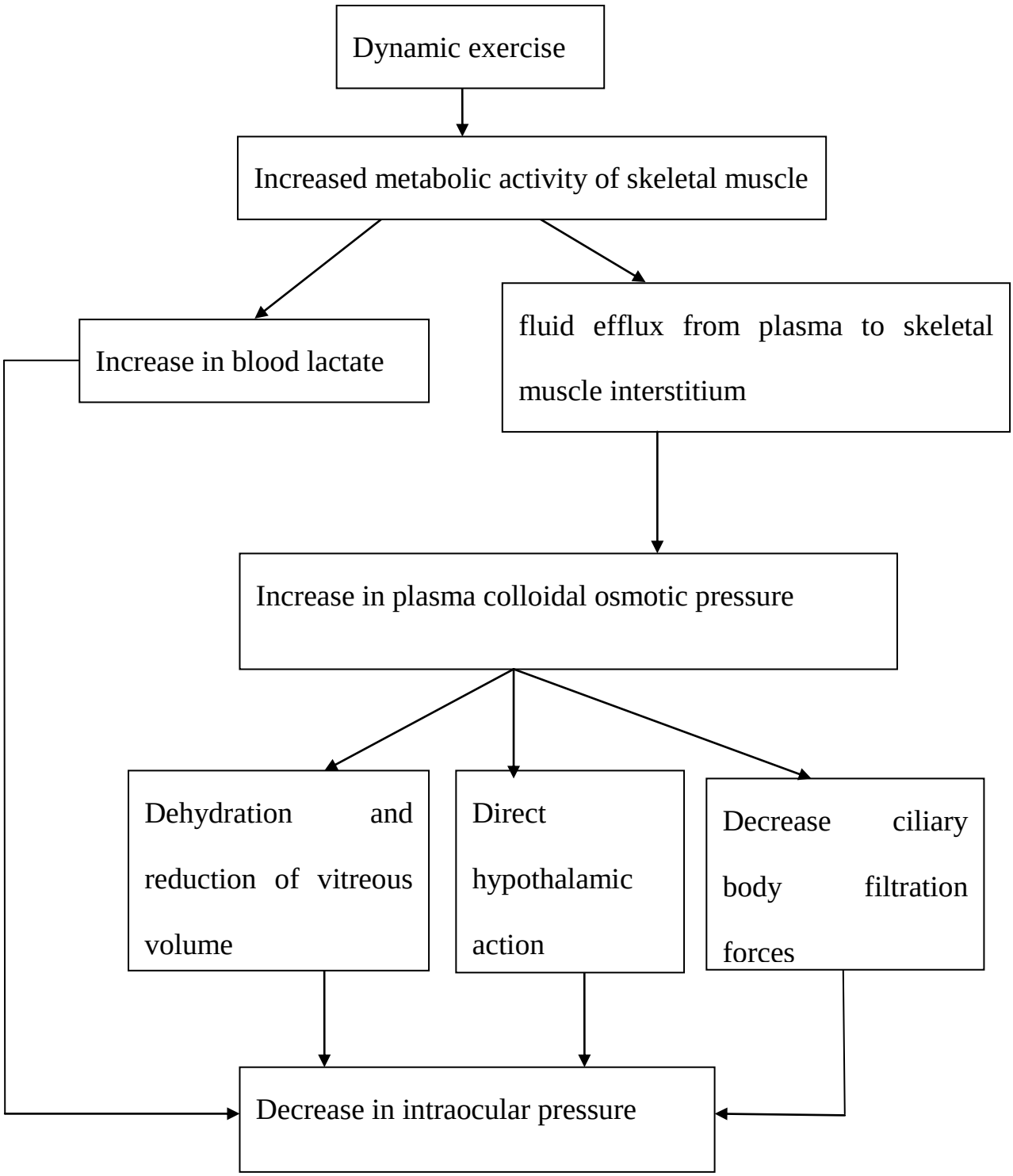
Supine position and whole body head down tilt position causes rise in intraocular pressure which necessitates the history of type of exercises done by the individual (yoga, physical training). Valsava maneuver, electroshock therapy and playing high resistant musical instruments causes increases in intraocular pressure.[61-67]

Exercise lowers intraocular pressure in patients with or without glaucoma. The effect of aerobic exercise on intraocular pressure lowering was observed in patients under glaucoma medications. Exercise basically divides in to isometric and isotonic exercise, static and dynamic exercise, aerobic and anaerobic exercise. In isometric exercise there will be increase in tension without shortening of length of the muscle. This type of exercise builds the muscle and increases the fitness of an individual. In isotonic exercise there will be changes in the muscle length with constant tension.

Intraocular pressure reduction is directly proportional to intensity of workload. Numerous physiological changes that occur with exercise have been implicated as possible mechanisms in the ocular hypotensive response to exercise. some of them are increase in plasma

osmolarity, increase in blood lactate levels, changes in PCO_2, changes in colloidal osmotic pressure, increased sympathetic activity, increased fibrinolytic activity of schlemm's canal. As lactic acid enters the general circulation blood lactate levels rise, which causes an increase in blood osmolarity and reduction in pH. The increase lactate levels cause an out flux of water from the eye which is responsible for fall in IOP with exercise. Stimulation of the sympathetic nervous system during exercise causes the release of large quantities of epinephrine and nor epinephrine from adrenal medulla. Epinephrine reduces IOP by lowering outflow resistance and by lowering the rate of aqueous formation. Hormones like epinephrine, norepinephrine, vasopressin, insulin, glucocorticoids, mineral corticoid, corticotrophin and growth hormone appear to increase aqueous out flow facility and decrease aqueous humour production.

Flow chart depicting the mechanism of intraocular pressure reduction during exercise:



7) EYELID AND EYE MOVEMENT:

Blinking causes rise in intraocular pressure by 10 mm of Hg. Eyelid squeezing causes a rise as high as 90 mm of Hg.[68] Voluntary eyelid fissure widening causes rise in intraocular pressure of 2 mm of Hg.[69] During strabismus surgery, particularly with thyroid ophthalmopathy intraocular pressure rises as much as 84 mm of Hg. [70]

8) SYSTEMIC CONDITIONS:

Diabetes , hypertension, obesity, graves disease, hyperthermia, sleep apnoea increases intraocular pressure. AIDS and myotonic dystrophy decreases it.[71]

V) TONOMETRY:

Intraocular pressure is measured by a technique known as tonometry. There are 2 types of tonometer. They are

1) Indentation tonometry

2) Applanation tonometry

INDENTATION TONOMETRY:

Indentation tonometry forms the base for schiotz tonometer which is used often for its simplicity and easy transportability. Schiotz developed the schiotz tonometer which was first designed to measure with relative reproducibility. The three parts of schitz tonometer are foot

plate, plunger and scale. The plunger is applied with weights of 5.5, 7.5, 10 and 12.5 mgs. The footplate is kept on the cornea and the cornea gets

Fig : 9 : Schiotz tonometer

indented by the plunger which causes the globe to get deformed. The scale which gets attached to the plunger measures this deformation which is converted to intraocular pressure by a nomogram known as friedenwald nomogram. The coefficient of ocular rigidity is given as constant K. it measures the resistance of eye to the distending force of tonometer.

LIMITATIONS:

K value is kept as average for all the eyes. so when the ocular rigidity is high or when it is getting low the K value becomes unreliable. Hypermetropia , chronic glaucoma and chronic vasoconstrictor therapy increases the ocular rigidity. Subsequently the intraocular pressure will be higher in these eyes. Myopia, miotic therapy, retinal detachment surgeries, intravitreal injection of gas and vasodilator therapy decreases the ocular rigidity thereby subsequently reduces the intraocular pressure. Scarred cornea, very thick corneas and steep corneas yields unreliable high false readings.

APPLANATION TONOMETRY:

Imbert ficks law is the basis for the applanation tonometry. It states that “in an ideal, dry , thin walled sphere, the pressure[P] equals the force needed to flatten the surface[F] divided by area of flattening [A]

P=F/A

Cornea is flattened about 3 mm in applanation tonometry in which 5 microlitre of fluid is minimally displaced. This flattening of 3mm alone supports the fact that scleral rigidity doesn't play a role in applanation tonometry.

A) SUBTYPES:

1) Variable area

2) Variable force

VARIABLE AREA:

These tonometers helps to measure the flattening area of cornea by a known amount of force.

Eg: maklakov tonometer

VARIABLE FORCE:

These tonometers helps to measure the force needed to flatten the standard area of cornea

Eg: goldmann applanation tonometer[current gold standard]

GOLDMANN APPLANATION TONOMETER:

This is mounted over a standard slit lamp. It has a plastic biprism which is mounted over a rod. After the cornea is anaesthetized, the sodium flourescein dye is instilled and the cobalt blue filter is switched on before touching the cornea. Two semicircles are seen when the observer views from the slit lamp. The knob of the tonometer is adjusted in such a way that the inner margins of both semicircles meet each other and start pulsating. At this point the readings are taken. The thickness of the circle plays a significant role because the thinner or thicker circle may produce false high or low readings

Other types:

1) Perkins tonometer

2) Pneumotonometer

3) Mackey-marg tonometer

4) Air puff tonometer

5) Dynamic contour tonometer

6) Rebound tonometer

7) Ocular response analyser

8) Non corneal transpalpebral tonometer [72]

GOLDMANN APPLANATION TONOMETER:

Fig:10: Goldmann applanation tonometer

VI) ETIOPATHOLOGICAL CONSIDERATIONS OF GLAUCOMA:

Progressive optic neuropathy due to death of retinal ganglion cells is the characteristic feature of all glaucomas. This leads to characteristic optic disc appearance and specific visual field defects.

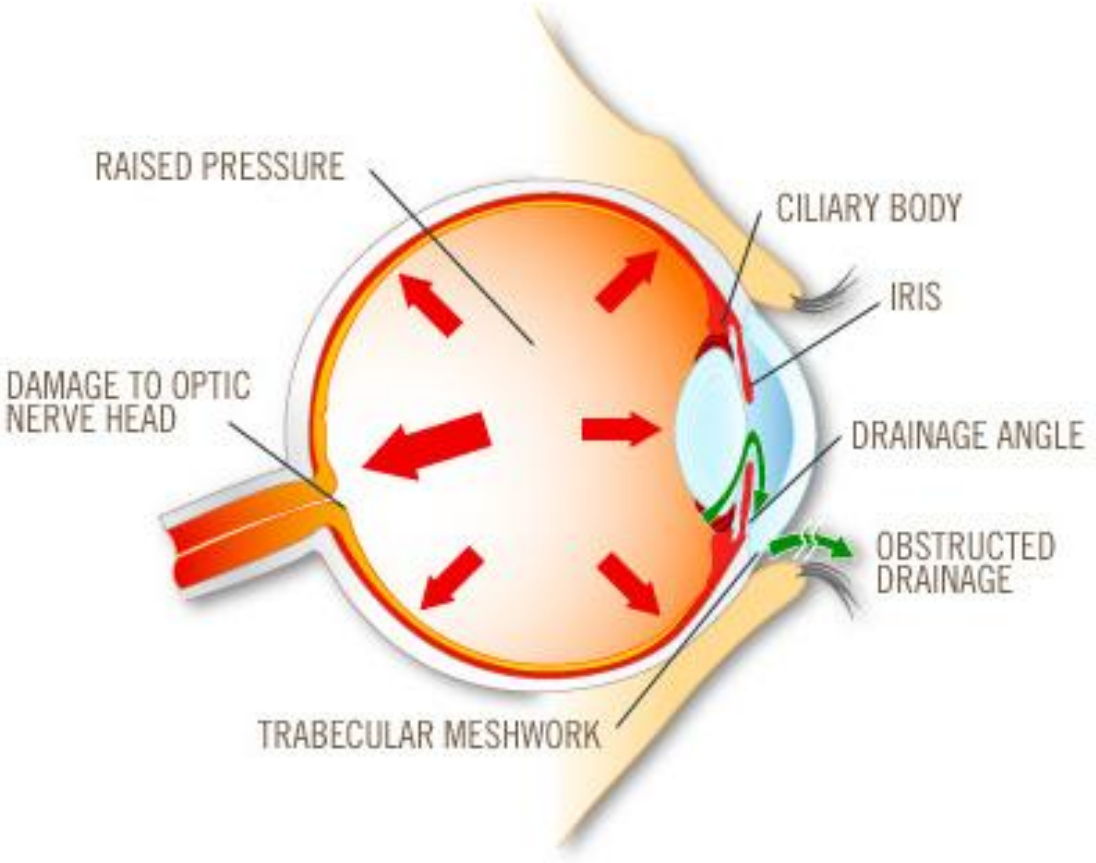


Fig:11: shows the pathogenesis of Glaucoma

VII) ETIOLOGY OF RGC DEATH:

A) Primary insult:

1) Raised IOP (mechanical theory)

2) Pressure independent factors (vascular insufficiency theory)

i) Failure of auto regulatory mechanism of blood flow.

ii) Vasospasm.(MIGRAINE, raynaud's phenomena)

iii) Systemic hypotension

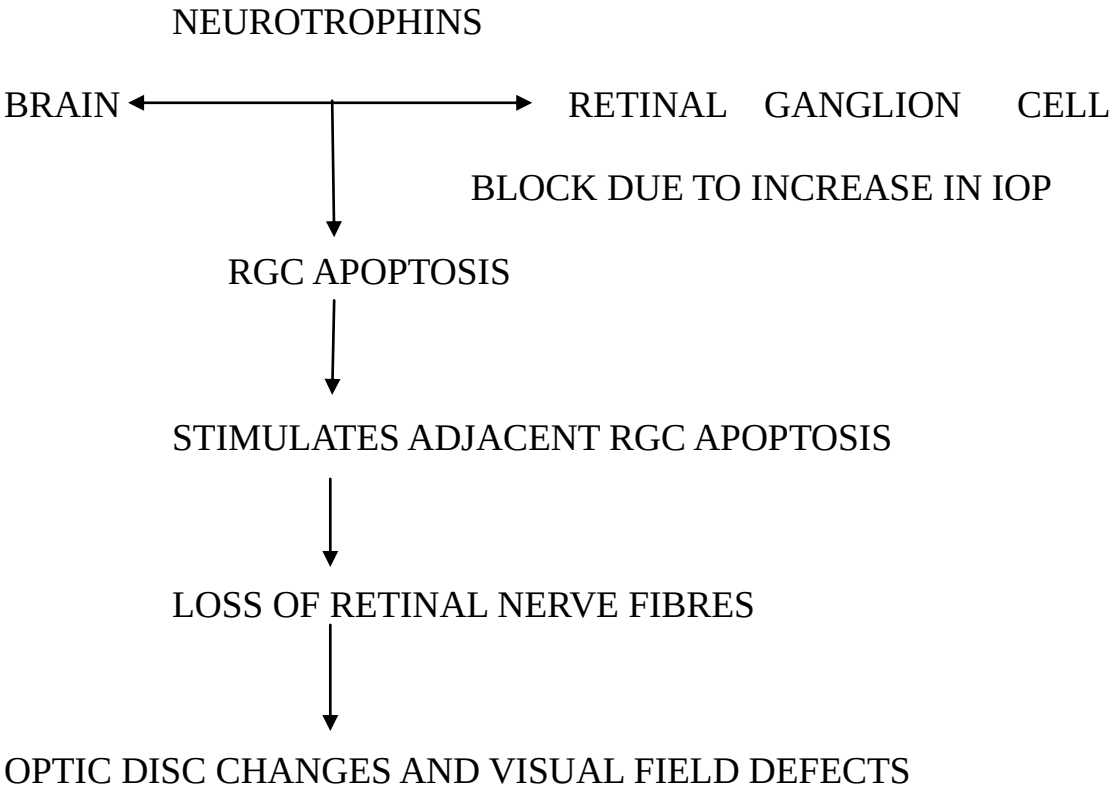
iv) Acute blood loss

v) Abnormal coagulative profile.

B) Secondary insult: (Excito toxicity theory)

Toxic factors released by RGC death due to primary insult.

VIII) PATHOGENESIS OF RGC DEATH:



NEUROTROPHINS:

Neurotrophins are the growth factors helping in the function of retinal ganglion cells, when there is pathological block in this transport from brain to retinal ganglion cells or from RGC to brain due to ischemia or so there will be apoptosis of retinal ganglion cells which inturn stimulates the adjacent RGC apoptosis with the loss of retinal nerve fibres and most importantly with the characteristic change in optic disc and visual field defect.

Glaucoma can be broadly classified in to open angle and angle closure glaucoma with the help of gonioscope.

XV. GONIOSCOPY:

Gonioscope is a special lens which is used to examine the angle of anterior chamber. The rays arising from the angle of anterior chamber are totally internally reflected because it strikes the corneal interface at more than 45 degrees which is more than the critical angle for this interface. Goniolens change the interface refracting properties and allows visualization of the angle.

INDICATIONS:

DIAGNOSTIC INDICATIONS:

1) Narrow peripheral anterior chamber
2) Evidence of angle closure
3) Evidence of inflammation
4) Evidence of neoplastic activity
5) Evidence of developmental anomaly
6) Classification of glaucoma
7) Extent of neovascularisation
8) Extent of angle recession
9) Planning of treatment in iris neovascularisation
10) Planning of treatment in laser procedures

THERAPEUTIC INDICATIONS:

1) Argon laser trabeculoplasty
2) Laser goniotomy
3) Reopening of trabecular opening
4) Opening of sclerostomy fistula

Angle of the anterior chamber can be assessed by two methods

1) Direct gonioscopes
2) Indirect gonioscopes

DIRECT GONIOSCOPES:

By using this goniolens the angle of anterior chamber can be viewed directly. the whole anterior chamber can be visualized in a panoramic view with the angle structures being placed in a normal spatial orientation. The above mentioned point is the most appreciable advantage of this type of goniolens.

(Eg)

1) koeppe: prototype
2) barkan: surgical
3) swan Jacob: for surgical use in children
4) layden: premature infants
5) Richardson Schaffer
6) Thorpe surgical goniolens

ADVANTAGES:

1) Simultaneous visualization of both eyes is possible
2) Good Binocularity
3) Distortion of the angle is minimal
4) The whole angle is viewed in a panoramic view
5) Examiner can easily manipulate the viewing angle

DISADVANTAGES:

1) Patient should be in a supine position
2) Operating microscopes are needed
3) Poor visualization of anatomical structures

INDIRECT GONIOSCOPE:

Indirect gonioscopy is a method in which Laterally inverted image of angle of anterior chamber is seen through a mirror. Three types of mirrors are commonly used. They are

1) Goldmann single mirror
2) Goldmann three mirror
3) Zeiss four mirror

ADVANTAGES:

1) The advantage of slit lamp magnification, controlled illumination and streopsis can be incorporated in this type of gonioscopy
2) Indentation and manipulation is achievable
3) Localization of angle structures is possible
4) Both the examiner and the patient are comfortable

DISADVANTAGES:

1) Reflected image is visualized
2) Inverted image and the opposite side angle are visualized
3) Distortion of the angle may be caused by the small aperture lens
4) Both the eyes cannot be compared simultaneously
5) Patient cooperation is inevitable

The differentiation between the true synechial angle closure and the appositional angle closure marks the important aspect of gonioscopy. There are two different methods to differentiate the true synechial angle closure from the appositional angle closure. They are

A) Manipulative gonioscopy
B) Indentation gonioscopy

MANIPULATIVE GONIOSCOPY:

The goldmann lenses whose diameter is greater than the corneal diameter are used in this method. The goniolenses are moved by the examiner superiorly if he want to visualize the superior angle in the inferior mirror orelse the patient is asked to look inferiorly. On examination ,If the viewing angle is opened up it is diagnosed as appositional angle closure orelse if the angle doesnot open, if it is closed it is labeled as synechial angle closure.

INDENTATION GONIOSCOPY:

The goniolenses whose diameter is smaller than that of the cornea are used in this method (Eg) four mirror lenses. In this method the lens is apposed towards the cornea. Subsequently the aqueous is pushed posteriorly in to the angle of the anterior chamber. On examination, the angle will open up if it is closed due to apposition.

The advantage of the above two techniques extend to the level that the iris processes and the peripheral anterior synechiae can be clearly differentiated. [73]

GRADING OF THE ANGLE OF ANTERIOR CHAMBER:

1) SPAETH'S GRADING:

A) LEVEL OF IRIS INSERTION:

- A (anterior to schwalbe's line)
- B (just behind schwalbe's line)
- C (at the scleral spur)
- D (deep angle CBB seen)
- E (extremely deep angle)

B)ANGULAR WIDTH- 10^{O} , 20^{o},30^{o},40^{o}

C) PERIPHERAL IRIS CONFIGURATION:

- q- queer
- r- regular
- s- steep

2) SHAFFER'S GRADING:

S.NO	GRADE	CONFIGURATION	ANGLE WIDTH	CHANCES OF CLOSURE	STRUCTURES VISIBLE ON GONIOSCOPY
1.	IV	WIDE OPEN	35-45	NIL	FROM SCHWALBE'S LINE TO CILIARY BODY
2.	III	OPEN ANGLE	20-35	NIL	FROM SCHWALBE'S LINE TO SCLERAL SPUR
3.	II	MODERATELY NARROW	20	POSSIBLE	FROM SCHWALBE'S LINE TO TRABECULAR MESHWORK
4.	I	VERY NARROW	10	HIGH	SCHWALBE'S LINE
5.	0	CLOSED	0	CLOSED	NONE OF THE ANGLE STRUCTURES VISIBLE

3) SCHEIE'S GRADING:

Reverse of shaffer's grading.

S.NO	GRADE	CONFIGURATION	ANGLE WIDTH	CHANCES OF CLOSURE	STRUCTURES VISIBLE ON GONIOSCOPY
1.	0	WIDE OPEN	35-45	NIL	FROM SCHWALBE'S LINE TO CILIARY BODY
2.	I	OPEN ANGLE	20-35	NIL	FROM SCHWALBE'S LINE TO SCLERAL SPUR
3.	II	MODERATELY NARROW	20	POSSIBLE	FROM SCHWALBE'S LINE TO TRABECULAR MESHWORK
4.	III	VERY NARROW	10	HIGH	SCHWALBE'S LINE
5.	IV	CLOSED	0	CLOSED	NONE OF THE ANGLE STRUCTURES VISIBLE

XVI. CLASSIFICATION:

S.NO	TYPES
1	Open-angle glaucomas without other known ocular or systemic disorders
2	Angle-closure glaucomas without other known ocular or systemic disorders
3	Developmental glaucomas
4	Glaucomas associated with other ocular and systemic disorders

OPEN-ANGLE GLAUCOMAS WITHOUT OTHER KNOWN OCULAR OR SYSTEMIC DISORDERS:

S.NO	SUBTYPES
1	Chronic open-angle glaucoma
2	Normal-tension glaucoma

ANGLE-CLOSURE GLAUCOMAS WITHOUT OTHER KNOWN OCULAR OR SYSTEMIC DISORDERS

S.NO	SUBTYPES
1	Pupillary block glaucomas
2	Combined mechanism glaucoma

DEVELOPMENTAL GLAUCOMAS

S.NO	SUBTYPES
1	Congenital glaucoma
2	Juvenile open-angle glaucoma (overlap with chronic open-angle glaucoma)
3	Axenfeld-Rieger syndrome
4	Peters anomaly
5	Aniridia
6	Other developmental anomalies

GLAUCOMAS ASSOCIATED WITH OTHER OCULAR AND SYSTEMIC DISORDERS

S.NO	SUBTYPES
1	Glaucomas associated with disorders of the corneal endothelium
2	Glaucomas associated with disorders of the iris and ciliary body
3	Glaucoma associated with disorders of the lens
4	Glaucomas associated with disorders of the retina, choroid, and vitreous
5	Glaucomas associated with intraocular tumors
6	Glaucomas associated with elevated episcleral venous pressure
7	Glaucomas associated with inflammation
8	Steroid-induced glaucoma
9	Glaucomas associated with ocular trauma
10	Glaucomas associated with hemorrhage
11	Glaucomas after intraocular surgery

GLAUCOMAS ASSOCIATED WITH DISORDERS OF THE CORNEAL ENDOTHELIUM

S.NO	SUBTYPES
1	Iridocorneal endothelial syndrome
2	Posterior polymorphous dystrophy
3	Fuchs endothelial corneal dystrophy

GLAUCOMAS ASSOCIATED WITH DISORDERS OF THE IRIS AND CILIARY BODY

S.NO	SUBTYPES
1	Pigmentary glaucoma
2	Iridoschisis
3	Plateau iris
4	Iris and ciliary body cysts

GLAUCOMA ASSOCIATED WITH DISORDERS OF THE LENS

S.NO	SUBTYPES
1	Exfoliation syndrome
2	Glaucomas associated with cataracts
3	Glaucomas associated with lens dislocation

GLAUCOMAS ASSOCIATED WITH DISORDERS OF THE RETINA, CHOROID, AND VITREOUS

S.NO	SUBTYPES
1	Neovascular glaucoma
2	Glaucomas associated with retinal detachment and vitreoretinal abnormalities

GLAUCOMAS ASSOCIATED WITH INFLAMMATION

S.NO	SUBTYPES
1	Glaucomas associated with uveitis
2	Glaucomas associated with keratitis, episcleritis, and scleritis

GLAUCOMAS AFTER INTRAOCULAR SURGERY

S.NO	SUBTYPES
1	Ciliary block (malignant) glaucoma
2	Glaucomas in pseudophakia and aphakia
3	Epithelial, fibrous, and endothelial proliferation
4	Glaucomas associated with corneal surgery
5	Glaucomas associated with vitreoretinal surgery

XVII. PRIMARY OPEN-ANGLE GLAUCOMA:

It is defined as a multifactorial optic neuropathy in which there is characteristic atrophy of the optic nerve. The following three criterias has to be fulfilled to call it is a typical primary open angle glaucoma :

(a) an intraocular pressure (IOP) consistently above 21 mm Hg in at least one eye.

(b) an open, normal-appearing anterior chamber angle with no apparent ocular or systemic abnormality that might account for the elevated IOP.

(c) typical optic nerve head damage and/or glaucomatous visual field damage.

The most prevalent type of glaucoma is primary open angle glaucoma and is also referred as chronic open-angle glaucoma (COAG), idiopathic open-angle glaucoma, chronic simple glaucoma, and open-angle glaucoma with damage.

SCREENING OF PATIENTS AT RISK:

COAG is called as “silent thief of sight” because the patient will become symptomatic only when 80 t0 90 % of the vision is lost. So as an ophthalmologist it is our prime duty to identify those patients who are at risk. The most significant and modifiable risk factor is increased intraocular pressure.

DOCUMENTED RISK FACTORS WITH RELATIVE RISK:

GOOD EVIDENCE

1) Age
2) Blacks vs whites
3) Family history
4) Myopia
5) Pseudoexfoliation
6) Diastolic perfusion pressure

FAIR EVIDENCE

1) Large C/D ratio
2) Diabetes mellitus
3) Optic disc hemorrhage

WEAK EVIDENCE

1) Systolic blood pressure
2) peripapillary atrophy
3) migraine (for NTG)
4) hypothyroidism
5) sleep apnea
6) autoimmune

CLASSIFICATION OF OPEN ANGLE GLAUCOMA

BASED ON OUTFLOW MECHANISMS

In gonioscopy, the anterior chamber angle structures (i.e, trabecular meshwork, scieral spur, and ciliary body band) are visible in the open-angle glaucoma.

The elements obstructing aqueous outflow may be located on the :

A) Anterior chamber side of the trabecular meshwork (pretrabecular mechanisms).
B) Within the trabeculum (trabecular mechanisms)
C) Distal to the meshwork, in Schlemm's canal

D) Further along the aqueous drainage system (posttrabecular Mechanisms).

A. PRETRABECULAR (MEMBRANE OVER GROWTH)

l. Fibrovascular membrane (neovascular glaucoma)
2. Endothelial layer, often with Descement-like membrane – (post penetrating glaucoma , posterior polymorphous dystrophy , irido corneal endothelial syndrome.)
3. Epithelial downgrowth
4. Fibrous ingrowth
5. Inflammatory membrane

B. TRABECULAR (OCCLUSION OF INTERTRABECULAR SPACES)

1. Idiopathic (COAG , steroid induced glaucoma)
2. Clogging of the trabecular meshwork (hemmorhagic and ghost cell glaucoma , hemolytic glaucoma, phacolytic glaucoma, melanomalytic glaucoma.)

3. Alterations of trabecular meshwork(edema, uveitis, scleritis , alkali burns, angle recession g;laucoma)
4. Neoplasia in the form of malignant tumours, neurofibromatosis, naevus of ota, juvenile xanthogranulomata .
5. Glaucoma associated with pigment in trabecular meshwork.(pigmentary glaucoma , pseudoexfoliation glaucoma)

C. POSTTRABECULAR

1. Obstruction of Schlemm's canal (collapse of canal, obstructed of canal by sickle RBC)

2. Elevated episcleral venous pressure (carotid cavernous fistula , Cavernous sinus thrombosis, retro bulbar tumours.)

CLINICAL FEATURES:

SYMPTOMS :

1) Asymptomatic
2) Mild head ache, eye ache
3) Defect in visual field which may be subjective
4) Presbyopic glasses changed frequently due to consistent pressure over the ciliary muscle
5) Delayed dark adaptation

SIGNS:

1) **Anterior segment:**

   i) Early stages – normal

   ii) Late stages – sluggish papillary reflex, haziness of cornea.

2) **IOP changes:**

   i) Early stages – normal with diurnal variation exaggerated (5mm hg → suspicious of glaucoma , 8 mm hg → diagnostic of glaucoma)

   ii) Late stages – >21 mm of Hg rise of IOP permanently

3) **Optic Disc changes:**

Fundus examination is mandatory to observe optic disc changes in POAG.

i) Early changes:

a) Optic cup becomes vertical oval

b) Asymmetric optic cup between two eyes> 0.2

c) Large cup

d) Splinter hemorrhages on the margin of optic disc

e) Retinal nerve fibre defect.

ii) Advanced changes:

a) Marked cupping

b) Neuroretinal rim thinning

c) Bayonetting sign

d) Pulsation of retinal arterioles

e) Lamellar dot sign

iii) Glaucomatous Optic atrophy:

Optic nerve head gets deeply excavated , appears white due to the loss of nerve tissue.

4) VISUAL FIELD DEFECTS:

It starts in the Bjerrum's area (10-25 degree of fixation) and intersects with the optic disc changes, then undergoes the following sequence:

i) Isoptre contraction

ii) Barring of blind spot

iii) Para central scotoma

iv) Seidles scotoma

v) Arcuate or Bjerrum ' s scotoma

vi) Ring or double arcuate scotoma

vii) Ronne's central nasal step

viii) Peripheral field defects

ix) Tubular vision

x) Temporal island of vision which is the last to get lost.

INVESTIGATIONS:

1) Tonometry(applanation and schitz tonometer)
2) Diurnal variation test
3) Gonioscopy
4) Documentation of optic disc changes:
   i) Serial drawings
   ii) Photography
   iii) Photogrammetry
   iv) Slit lamp examination of anterior segment
   v) Perimetry
   vi) Nerve fibre layer analyser
   vii) Provocative tests (water drinking test, combined water drinking and tonography, bulbar pressure test, pres coline test and caffeine test.

XVIII. NORMAL TENSION GLAUCOMA:

It is also called as Low tension glaucoma and is associated with

1) Glaucomatous disc changes
2) Visual field defects
3) IOP constantly < 21 mm Hg.

XIX. OCULAR HYPERTENSION:

It is also called as Glaucoma Suspect and associated with IOP always greater than 21 mm Hg without optic disc changes and visual field defects.

XX. ANGLE CLOSURE GLAUCOMA:

It is classified in to three types. They are

1) Primary angle closure suspect
2) Prmary angle closure
3) Primary angle closure glaucoma[(1)]

AIM :

To study the effect of Exercise (treadmill) on Intraocular pressure

REVIEW OF LITERATURE :

Ashkenazi et al studied the effect of a continuous 110-km march with a 20-kg backpack load on intraocular pressure (IOP), plasma osmolarity, blood lactate, pH, and other related laboratory parameters in 22 healthy young volunteers. Intraocular pressure decreased significantly at all marching intervals and returned to baseline level 3 hr after the completion of marching. The maximal average reduction during marching was 4.1 mmHg. There was no correlation between IOP changes and levels of pH, blood lactate, serum proteins, and electrolytes or hematologic parameters. But IOP was found to be inversely related to plasma osmolarity during & after strenuous exercise.[73]

Tham CC in his study (2010) revealed that there is a acute but transient reduction in IOP after exercise , inspite there are exclusions like pigmentary glaucoma and weight lifting. He emphasises that exercise brings good health to human in all aspects , so exercise can be included as a factor for reducing IOP. [74]

Lipkova J et al. (2008) in his study " The Effect of Aerobic Exercises on the Ocular Parameters and the Psychic State of Glaucoma

Patients" conducted on 15 glaucoma patients of mean age 49 years. Eight of them enrolled in the aerobic exercise program and 7 of them serve as controls without any organized physical activity. Women who were in the study group done exercise 3 times a week for 45 mins for a period of 8 weeks. Goldmann applanation tonometer and schiotz tonometer were used for measuring IOP. Quantification of visual field defects was done by Octopus glaucoma program and psychical state was evaluated by EPI (Eysenck Personality Inventory) and STAI (State and Trait Anxiety inventory). From the study, they inferred that there was no effect on IOP by doing aerobic exercises in acute manner.[75]

Brownlee P et al. analysed the effect of moderate exercise on intraocular pressure and ocular blood flow(2004) in 31 subjects(20 males and 11 females). Intraocular pressure and pulsatile ocular blood flow were measured by ocular blood flow analyser. They concluded that there was significant reduction in IOP and increase in pulsatile ocular blood flow after moderate exercise like riding a stationary bicycle for 10 minutes for 3-5 times a week. The mean average reduction in intraocular pressure is 4.4 mm of Hg.[76]

A clinical study done by Mohammed Ehtesham et al revealed that there was a significant decrease in iop in group with BMI greater than 22 than in group with BMI less than 22.[77]

Sunitha et al studied the effect of isotonic exercise on iop. IOP was measured before and after exercise in 35 male subjects with no ocular abnormality with ages ranging from 18-25 years. Harvard step test was used for isotonic exercise till exhaustion. IOP was recorded immediately, 5 minutes and 10 minutes after exercise. The IOP showed a statistically significant fall following exercise. The mean IOP significantly decreased ($p<0.05$), immediately and after 5 minutes of isotonic exercise but drop in iop was not significant at 10 minutes.[78]

A clinical study done by Marcus et al in 1970 revealed that after four minutes walking in treadmill there was a significant decrease in intraocular pressure, blood pH, increase in serum osmolarity and blood lactate. In their study, 12 volunteers with no ocular pathology were included. Intraocular pressure, blood pressure, pulse rate and blood samples were taken in the fasting state before and immediately after exercise and at 15,30 and 60 minutes after exercise. Goldmann applanation tonometer was used in this study. The average reduction in intraocular pressure (5.9 mm Hg) immediately after exercise was highly significant ($p < 0.001$) and the intraocular pressure returned to baseline after 60 minutes.[79]

A study in 2009 "Aerobic exercise and intraocular pressure in normotensive and glaucoma patients" done by Natsis k et al suggests that there is a statistically significant reduction in IOP during jogging regardless of the usage of b-blocker or a prostaglandin analogue or an α-agonist in both normotensives and glaucoma patients. The mean reduction in intraocular pressure in both healthy individuals and glaucoma patients were 2.13 ± 0.72 and 2.6 ± 0.81 mm of Hg respectively. [80]

Vieria GM et al in 2002 studied the acute effects of resistance exercise on intraocular pressure and found that there was a small but significant reduction in intraocular pressure in 25 healthy volunteers after resistance exercises that is after lifting about 85% top load for eight times. The mean reduction in intraocular pressure is 1.61 mm of Hg.[81]

"Effects of exercise on ocular health" a study conducted by Hilton E in 2003 suggests that there is significant reduction in IOP after doing exercise along with other hemodynamic benefits. The reduction in intraocular pressure is mainly because of ocular dehydration which occurs due to changes in retinal and uveal vasculature, alteration in colloidal osmotic pressure which may directly reduce aqueous formation

or act on hypothalamus to reduce aqueous secretion through an unspecified response.[82]

A study was done by Price et al in june 2003 to assess the Effect of Exercise on Intraocular Pressure and Pulsatile Ocular Blood Flow in a Young Normal Population. 18 subjects with no ocular pathology were subjected to 4-min period of bicycle ergometry. Intraocular pressure and pulsatile ocular blood flow were measured before and immediately after exercise and at 5,10,20 and 30 minutes after exercise. There was significant reduction in intraocular pressure which returned to baseline over 30 minutes and increase in pulsatile ocular blood flow which returned to baseline within 10 minutes after stopping exercise. The average reduction in intraocular pressure was 3.82 mm of Hg.[83]

Qureshi IA(1996) in his case control study "Effects of exercise on intraocular pressure in physically fit subjects" suggests that 3 months duration of supervised exercise programme has reduced the IOP considerably. The average reduction in intraocular pressure were around 4 mm of Hg.[84]

"Effects of exercise on intraocular pressure and ocular blood flow: a review" by Risner D (2009) et al states that dynamic exercises

causes acute reduction in intraocular pressure which returns to pretrained levels after 1 month of cessation of exercise.[85]

Read et al studied the effect of short term influence of exercise on iop and axial length. 20 young adult subjects participitated for whom Baseline measures of ocular biometrics, IOP and ocular pulse amplitude (OPA) were taken. Measures of ocular biometrics, IOP and OPA were repeated before and after exercise. Exercise resulted in significant reductions in iop,ocular pulse amplitude and decrese in axial length.[86]

Qureshi et al studied the effects of sitting, walking, jogging, and running fast till exhaustion on the IOP of 15 healthy sedentary male volunteers. Intraocular pressures were measured with a Goldmann applanation tonometer before and after exercise. Post-exercise measurements were taken after every 10 minutes until IOP returned to pre-exercise levels. The effects of all tests were similar on both eyes. It is concluded that all forms of exertion decrease IOP. The mean reduction in intraocular pressure after sitting, walking, jogging and running were 1.20 ± 0·66, 3·20 ± 1·19, 5·07 ± 1·76 mmHg and 5·7 ± 1·09 mmHg respectively.[87]

“The Impact of Acute Dynamic Exercise on Intraocular Pressure:role of beta2 Adrenergic Receptor Polymorphism “ a study in

2002 by Gungor K et al suggests that B2AR gene is responsible for exercise induced decrease in IOP and he leaves way for future analysis of B2AR gene polymorphism for treating glaucoma.[88]

A research conducted by Qureshi IA in 1996 “Magnitude of decrease in IOP depends upon the intensity of exercise” reveals that in 25 healthy male sedentary lifestyle individuals, the acute initial decrease in intraocular pressure depends on the intensity of the exercise and not on the duration, quantity of the exercise, blood pressures or body mass index. In his study the mean reduction in intraocular pressure after exercising 7.5 minutes at 80% HRmax, 10 minutes at 60% HR max and 30 minutes at 40% HRmax were 4.7±0.9, 3.5±0.7 and 0.9±0.4 mm of Hg respectively.[89]

In 24 normotensive Nigerian individuals after 10 minutes of exercise there was a statistically significant reduction in intraocular pressure which lasts for 20 to 40 minutes. Then after 50 minutes there was a slight rise in IOP. This stresses the importance of exercise as a form of therapy which can be combined with anti glaucoma drugs in the management of glaucoma. The mean reduction in intraocular pressure was 2.17 mm of Hg.[90]

Prevention of Glaucoma through Exercise: A meta-analysis conducted by Gabrielle Roddy and Dave Ellemberg in 2012 states that mild exercise decreases IOP significantly whereas the role of moderate and severe forms of exercise in decreasing the IOP is insignificant.[91]

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In one study that included 7 subjects with glaucoma and 7 subjects without glaucoma, the IOP in all subjects dropped between 56 to 61%, following five minutes of walking and jogging.[92]

The effect of exercise may dissipate once subjects stop exercising. In a study of 9 sedentary glaucoma suspects who underwent 3 months of aerobic training, IOP levels returned to their original measurements three weeks after exercise cessation. Overall, exercise seems to have a beneficial effect on IOP.[93]

INCLUSION CRITERIA:

Medical students in the age group of 18-25 years

EXCLUSION CRITERIA:

- Any history of systemic diseases, smoking, ocular diseases[glaucoma etc], use of any systemic or local medications, any nutritive supplements, any psychiatric disorders, people doing Yoga, who had undergone yoga or eye surgery within last 3 months were excluded

- Subjects with cardiac diseases, respiratory diseases & physical deformity in legs were excluded

MATERIALS AND METHODS :

This prospective study involved measurement of the intraocular pressure using the Goldmann applanation tonometer in 46 medical students of PSG IMS&R aged between 18 to 25 years after getting the institutional ethical approval and consent from all the participants. No conflict of interest was there. After explaining the procedures which starts by the application of anaesthetic (Aurocaine) eye drops and instillation of flourescein dye, pre exercise intraocular pressure was measured using goldmann applanation tonometer, then the subject is asked to walk on the treadmill for 30 minutes with the speed of 7 km/hour. Then the subject's intraocular pressure is measured after exercise using the same method.

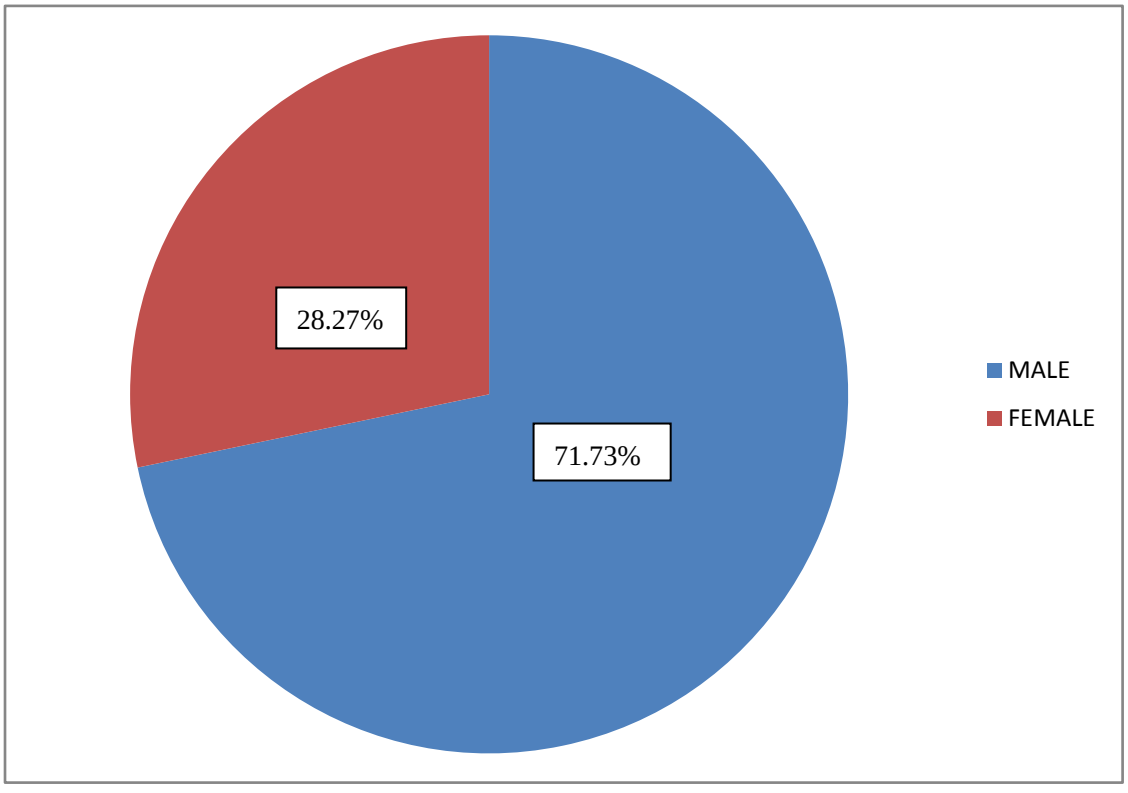
RESULTS :

SAMPLE SIZE (n=46)

TOTAL NUMBER OF MALES = 33

TOTAL NUMBER OF FEMALES= 13

Pie chart 1:



RIGHT EYE

The minimum, maximum ,mean and standard deviation of right eye pre exercise intra ocular pressure in both males and females are 8 mm Hg, 18 mm Hg, 14.43 mm Hg and 2.5 respectively and the minimum, maximum ,mean and standard deviation of post exercise intraocular pressure in both males and females are 8 mm Hg, 16 mm Hg, 11.13 mm Hg and 1.92 respectively.

(Table – 1)

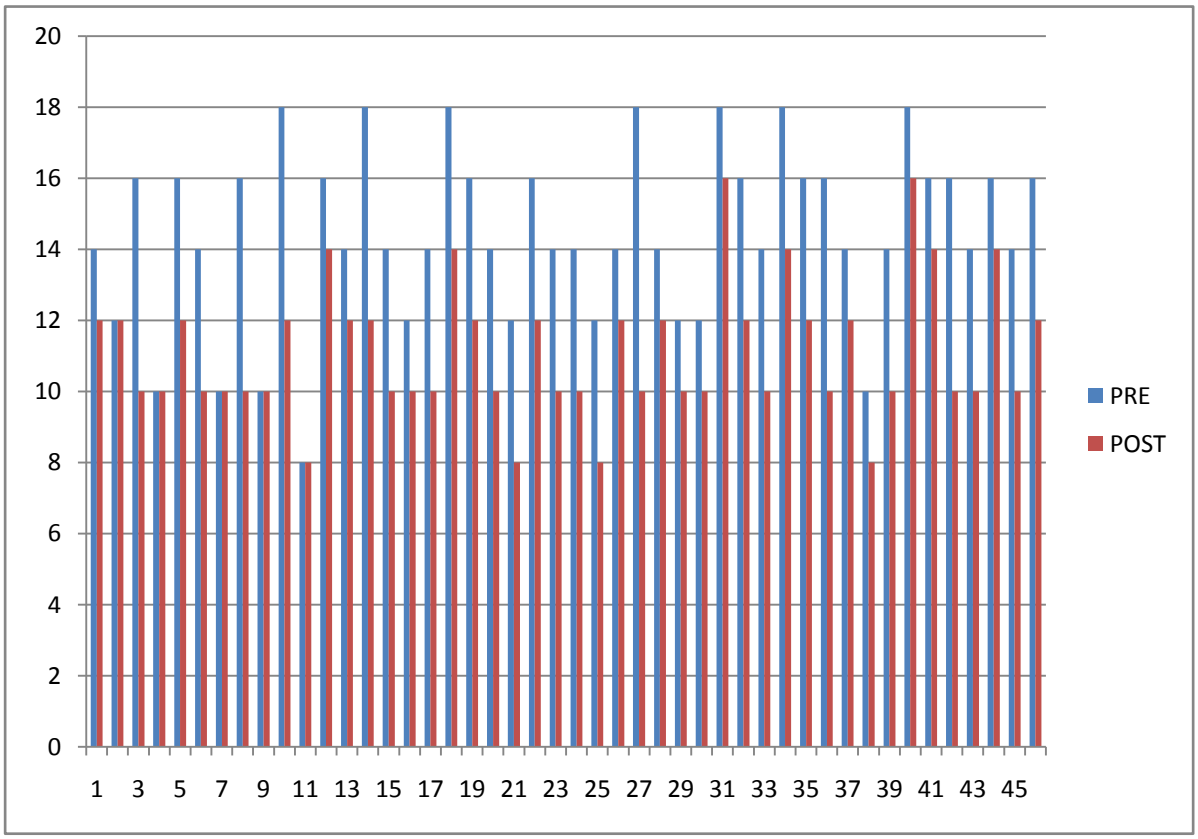
Table - 1:

S.NO	VALUES	RIGHT EYE (IOP) (MM OF HG)	
		PRE EXERCISE	POST EXERCISE
1	MINIMUM	8	8
2	MAXIMUM	18	16
3	MEAN	14.43	11.13
4	STANDARD DEVIATION	2.5	1.92

RIGHT EYE

HISTOGRAM SHOWING PRE AND POST EXERCISE INTRAOCULAR PRESSURE CHANGES IN BOTH MALES AND FEMALES:

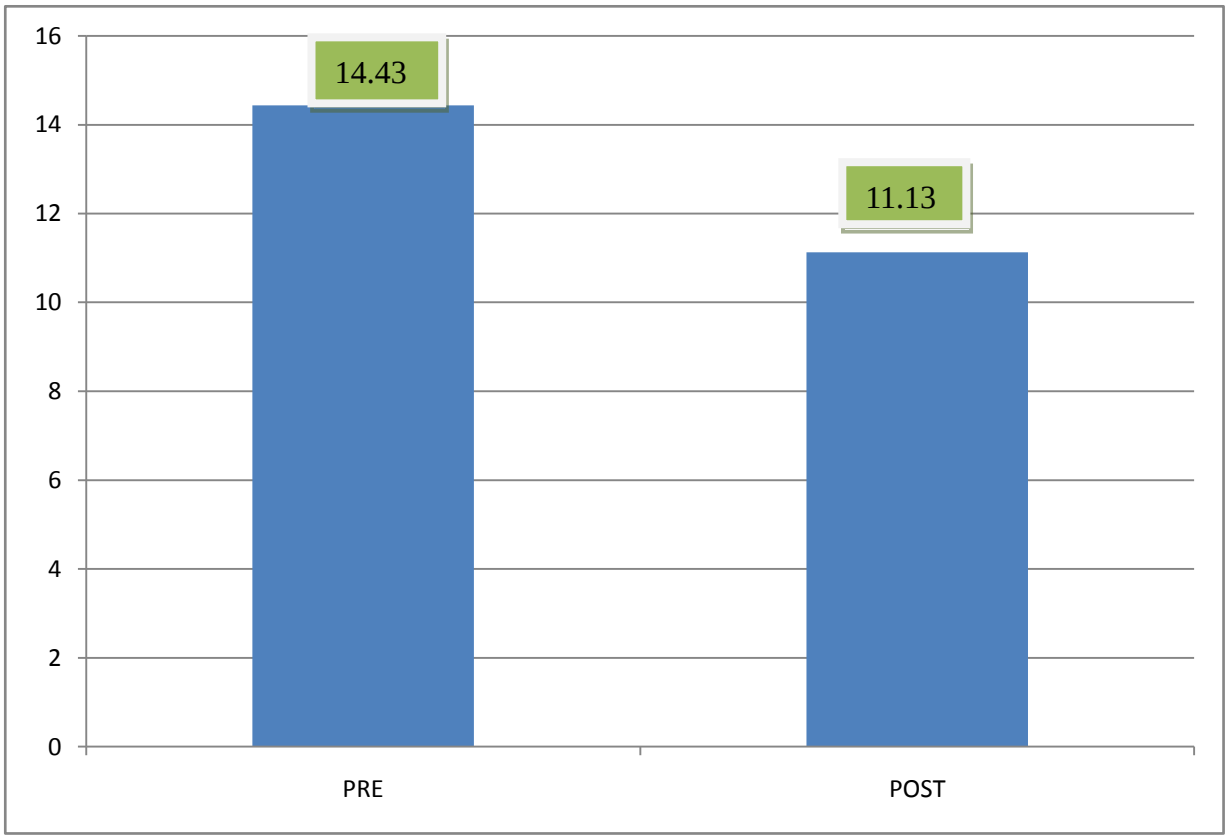
Histogram 1:



RIGHT EYE

HISTOGRAM COMPARING THE MEAN VALUE OF PRE AND POST EXERCISE INTRAOCULAR PRESSURE IN BOTH MALES AND FEMALES:

Histogram 2:



LEFT EYE

The minimum, maximum ,mean and standard deviation of left eye pre exercise intra ocular pressure in both males and females are 10 mm Hg, 18 mm Hg, 14.48 mm Hg and 2.61 respectively and the minimum, maximum ,mean and standard deviation of post exercise intraocular pressure in both males and females are 8 mm Hg, 14 mm Hg, 11 mm Hg and 1.73 respectively.

(Table – 2)

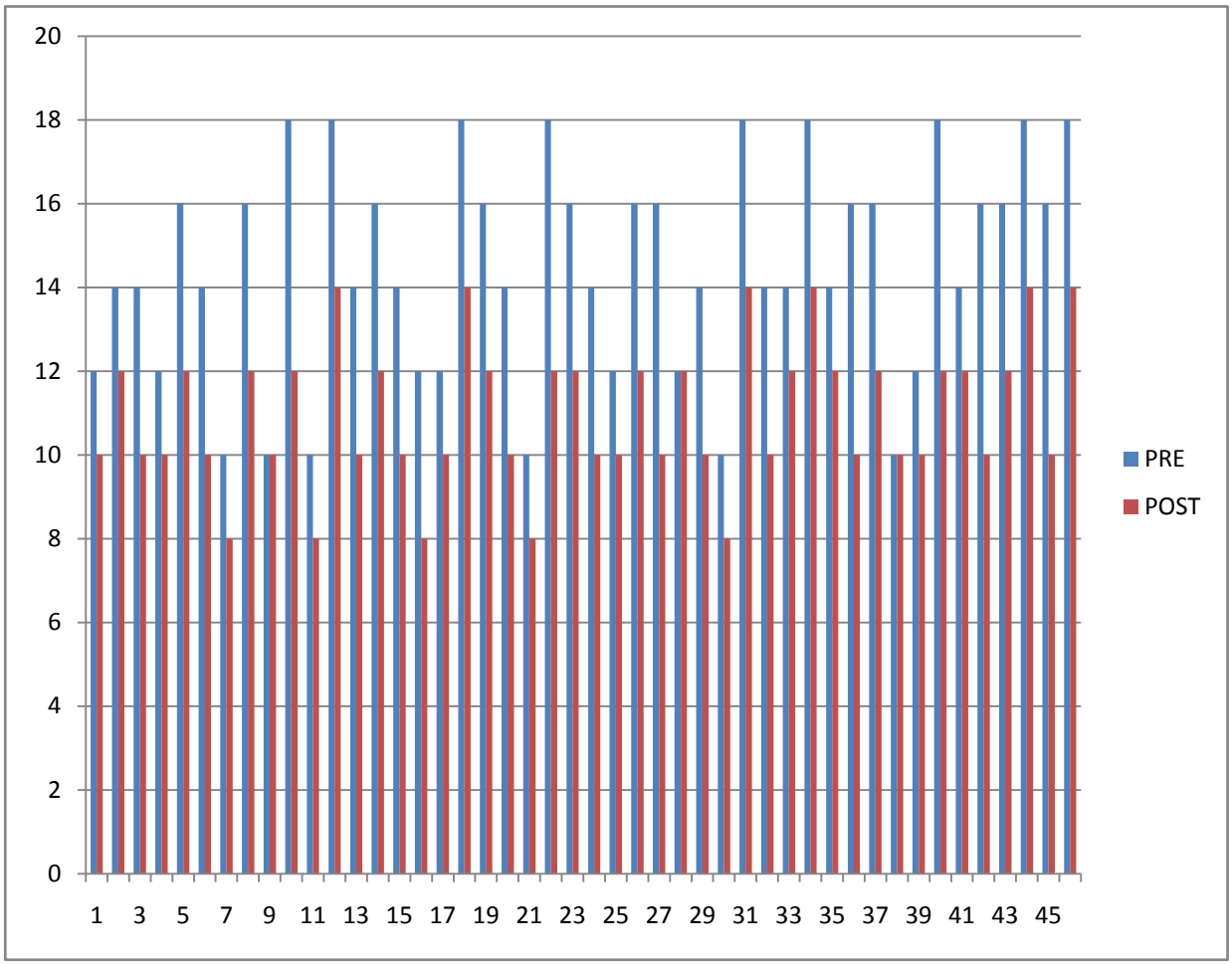
TABLE - 2

S.NO	VALUES	LEFT EYE (IOP) (MM OF HG)	
		PRE EXERCISE	POST EXERCISE
1	MINIMUM	10	8
2	MAXIMUM	18	14
3	MEAN	14.48	11.00
4	STANDARD DEVIATION	2.61	1.73

LEFT EYE

HISTOGRAM SHOWING PRE AND POST EXERCISE INTRAOCULAR PRESSURE CHANGES IN BOTH MALES AND FEMALES:

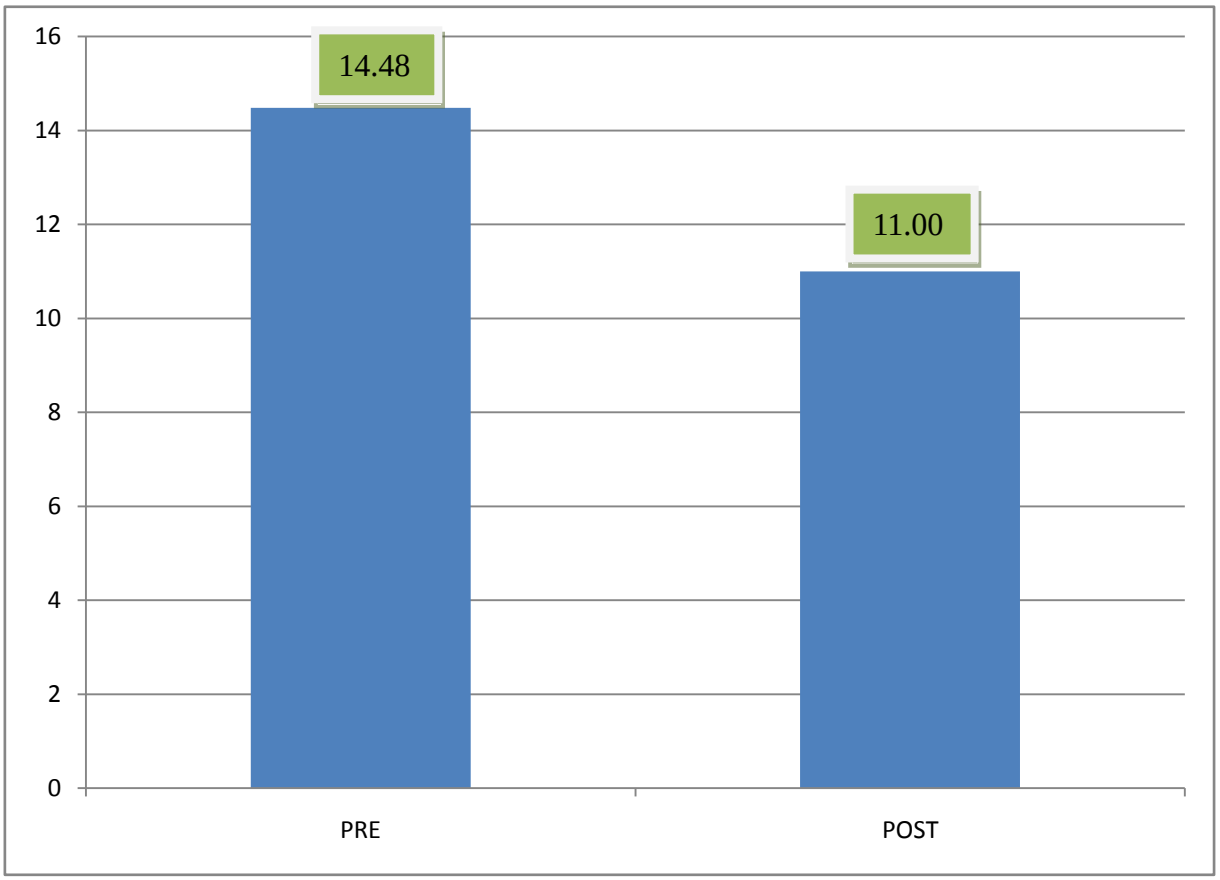
Histogram 3:



LEFT EYE

HISTOGRAM COMPARING THE MEAN VALUE OF PRE AND POST EXERCISE INTRAOCULAR PRESSURE IN BOTH MALES AND FEMALES:

Histogram 4:



RIGHT EYE

The minimum, maximum ,mean and standard deviation of right eye pre exercise intraocular pressure in males are 8 mm Hg, 18 mm Hg, 14.12 mm Hg and 2.56 respectively and the minimum, maximum ,mean and standard deviation of post exercise intraocular pressure in males are 8 mm Hg, 16 mm Hg, 10.91 mm Hg and 1.74 respectively.

(Table – 3)

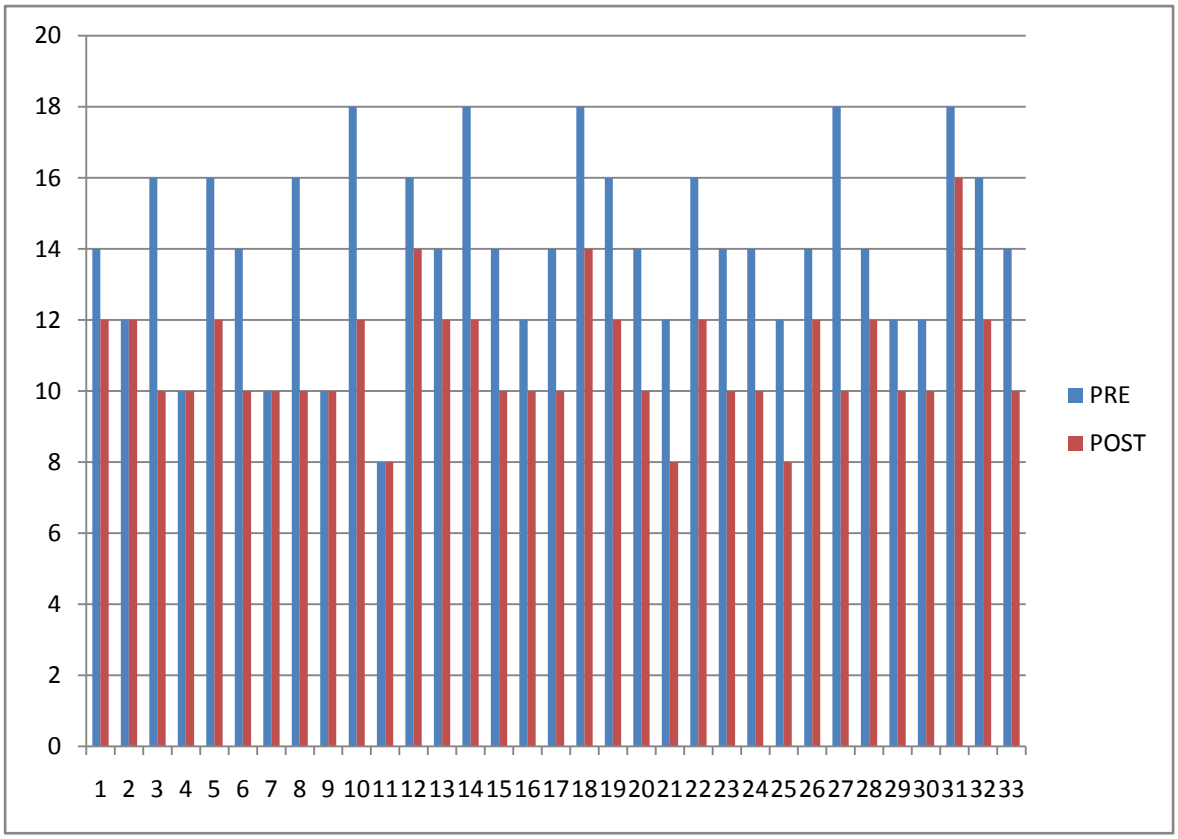
TABLE - 3

S.NO	VALUES	RIGHT EYE (IOP) (MM OF HG)	
		PRE EXERCISE	POST EXERCISE
1	MINIMUM	8	8
2	MAXIMUM	18	16
3	MEAN	14.12	10.91
4	STANDARD DEVIATION	2.56	1.74

RIGHT EYE

HISTOGRAM SHOWING PRE AND POST EXERCISE INTRAOCULAR PRESSURE CHANGES IN MALES:

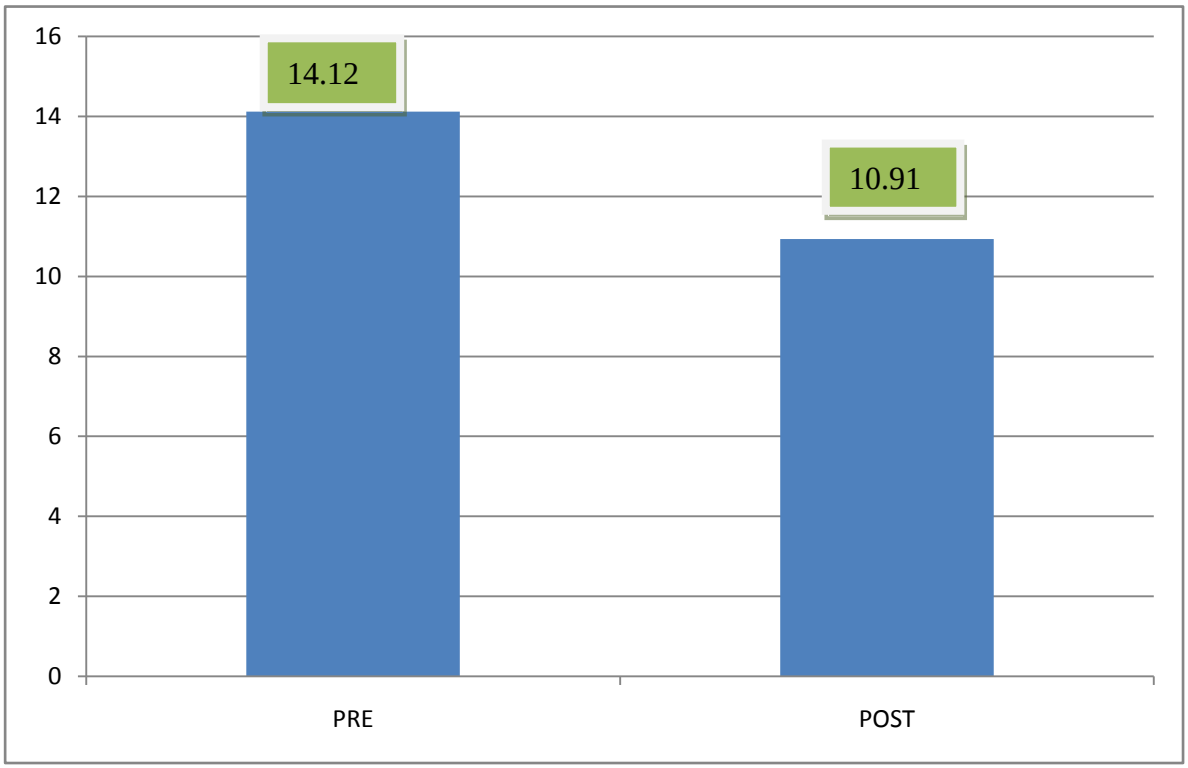
Histogram 5:



RIGHT EYE

HISTOGRAM COMPARING THE MEAN VALUE OF PRE AND POST EXERCISE INTRAOCULAR PRESSURE IN MALES:

Histogram 6:



LEFT EYE

The minimum, maximum ,mean and standard deviation of left eye pre exercise intraocular pressure in males are 10 mm Hg, 18 mm Hg, 14.10 mm Hg and 2.57 respectively and the minimum, maximum ,mean and standard deviation of left eye post exercise intraocular pressure in males are 8 mm Hg, 14 mm Hg, 10.73 mm Hg and 1.72 respectively. (table-4)

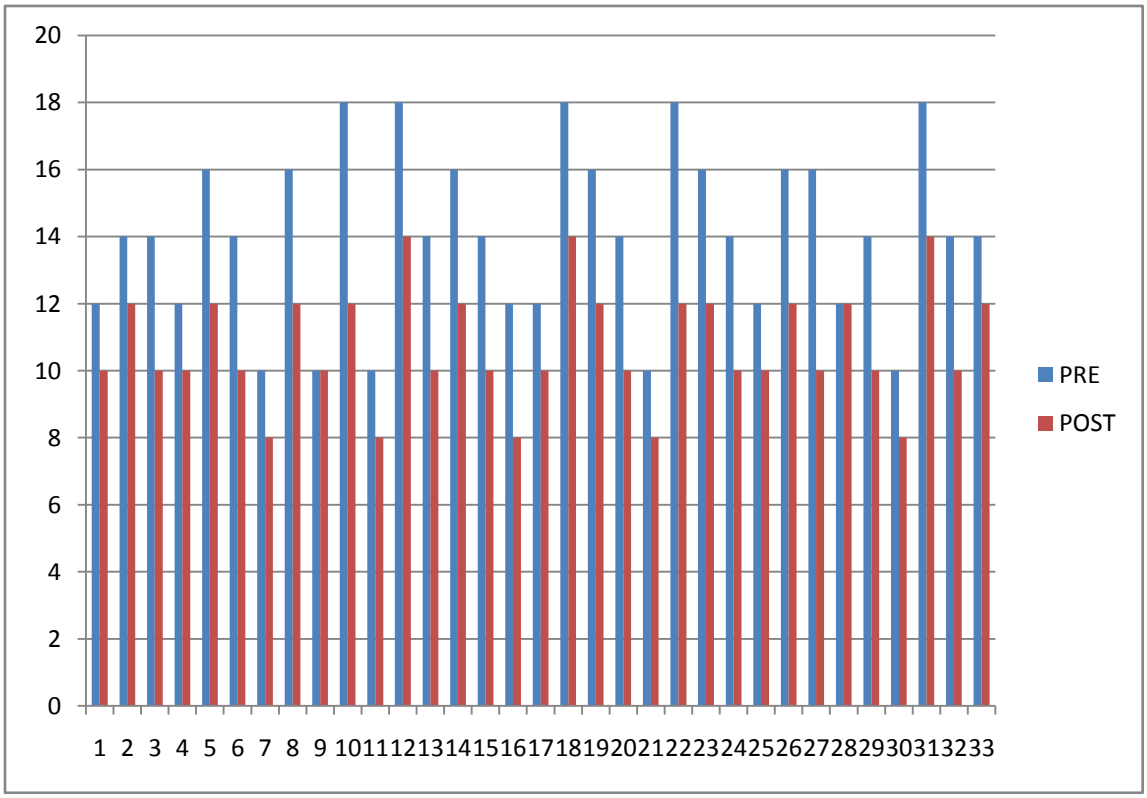
TABLE - 4

S.NO	VALUES	LEFT EYE (IOP) (MM OF HG)	
		PRE EXERCISE	POST EXERCISE
1	MINIMUM	10	8
2	MAXIMUM	18	14
3	MEAN	14.10	10.73
4	STANDARD DEVIATION	2.57	1.72

LEFT EYE

HISTOGRAM SHOWING PRE AND POST EXERCISE INTRAOCULAR PRESSURE CHANGES IN MALES:

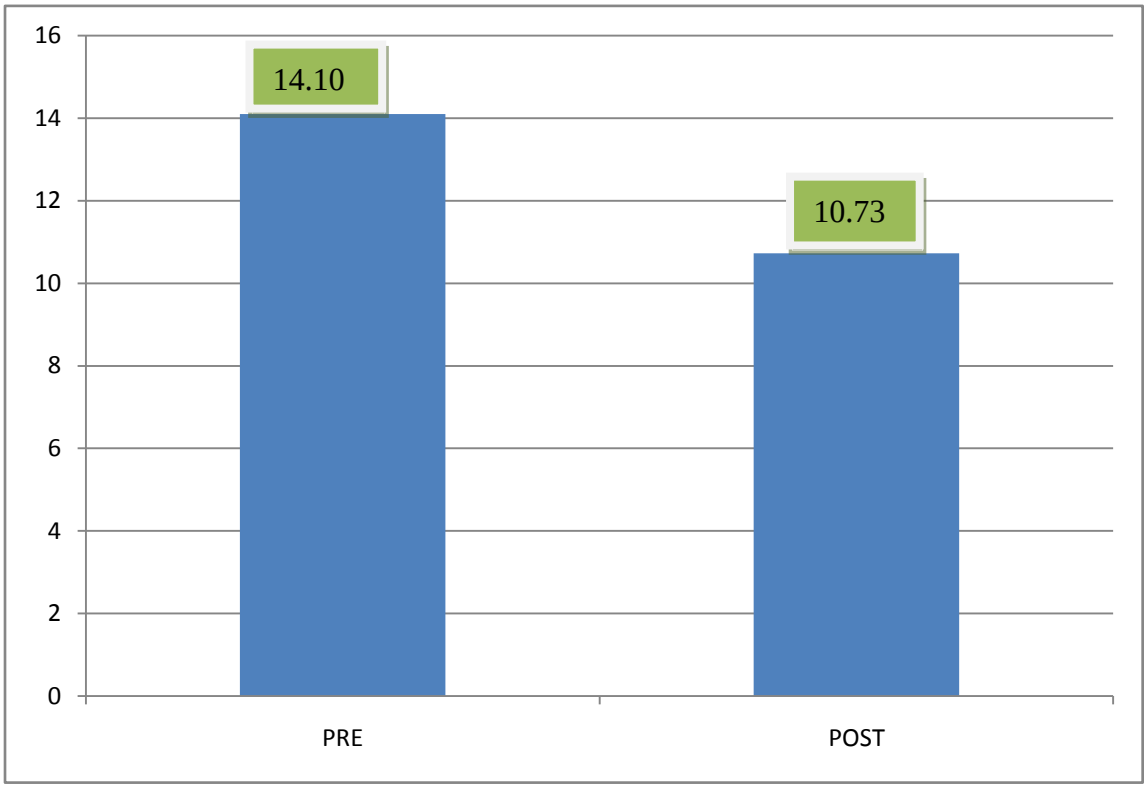
Histogram 7:



LEFT EYE

HISTOGRAM COMPARING THE MEAN VALUE OF PRE AND POST EXERCISE INTRAOCULAR PRESSURE IN MALES:

Histogram 8:



RIGHT EYE

The minimum, maximum ,mean and standard deviation of right eye pre exercise intraocular pressure in females are 10 mm Hg, 18 15.23 mm Hg, mm Hg and 2.10 respectively and the minimum, maximum ,mean and standard deviation of post exercise intraocular pressure in females are 8 mm Hg, 16 mm Hg, 11.69 mm Hg and 2.29 respectively. (table- 5)

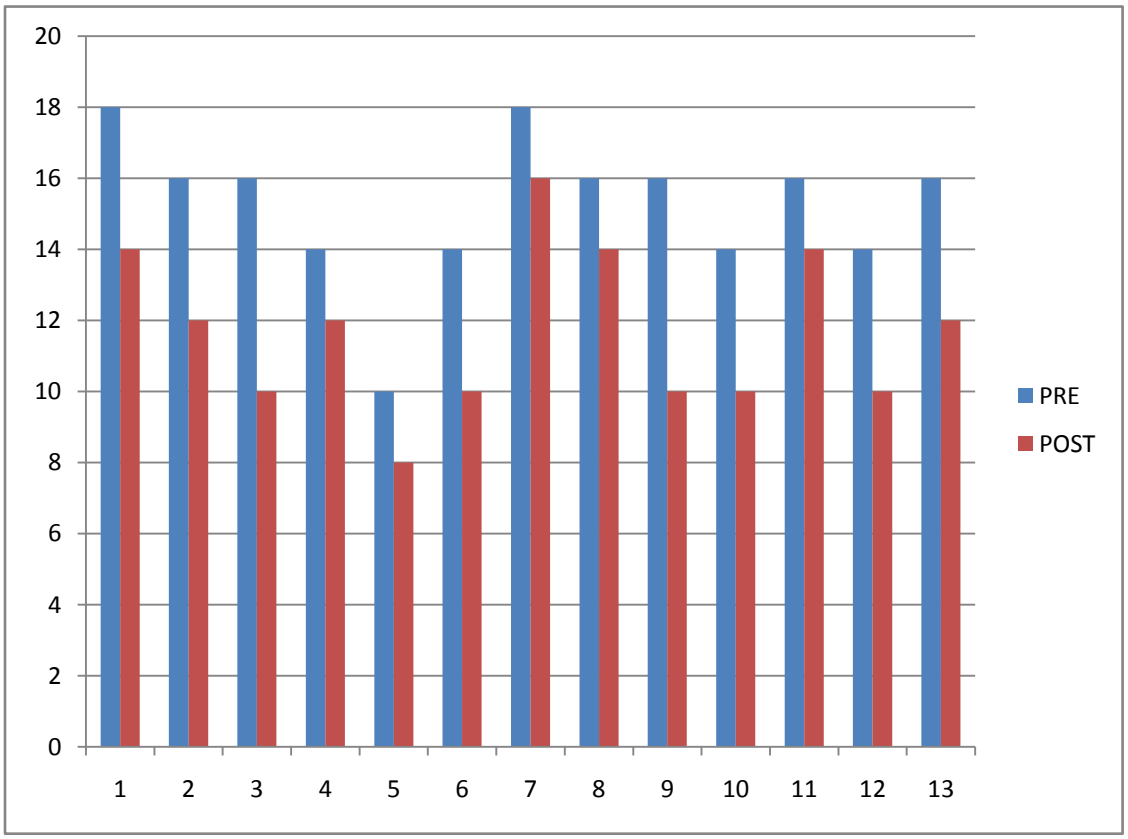
TABLE - 5

S.NO	VALUES	RIGHT EYE (IOP) (MM OF HG)	
		PRE EXERCISE	POST EXERCISE
1	MINIMUM	10	8
2	MAXIMUM	18	16
3	MEAN	15.23	11.69
4	STANDARD DEVIATION	2.10	2.29

RIGHT EYE

HISTOGRAM SHOWING PRE AND POST EXERCISE INTRAOCULAR PRESSURE CHANGES IN FEMALES:

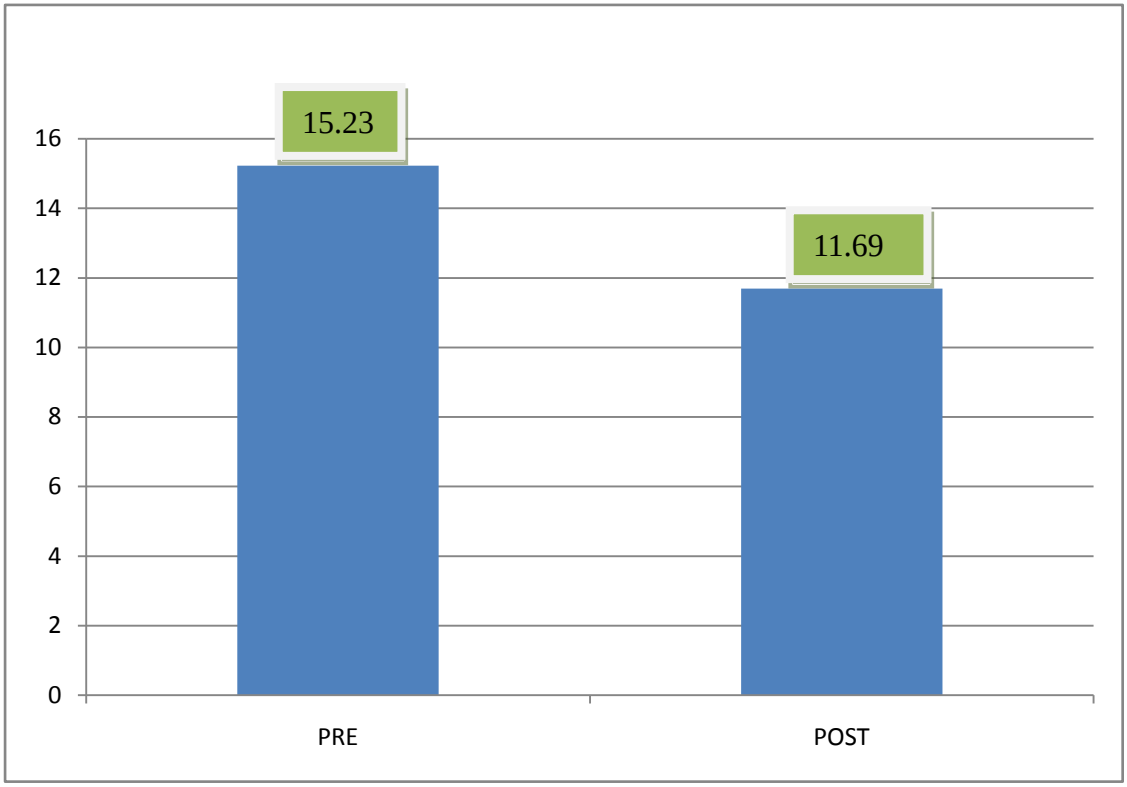
Histogram 9:



RIGHT EYE

HISTOGRAM COMPARING THE MEAN VALUE OF PRE AND POST EXERCISE INTRAOCULAR PRESSURE IN FEMALES:

Histogram 10:



LEFT EYE

The minimum, maximum ,mean and standard deviation of left eye pre exercise intraocular pressure in males are 10 mm Hg, 18 mm Hg, 15.54 mm Hg and 2.47 respectively and the minimum, maximum ,mean and standard deviation of post exercise intraocular pressure in males are 10 mm Hg, 14 mm Hg, 11.69 mm Hg and 1.60 respectively. (table – 6)

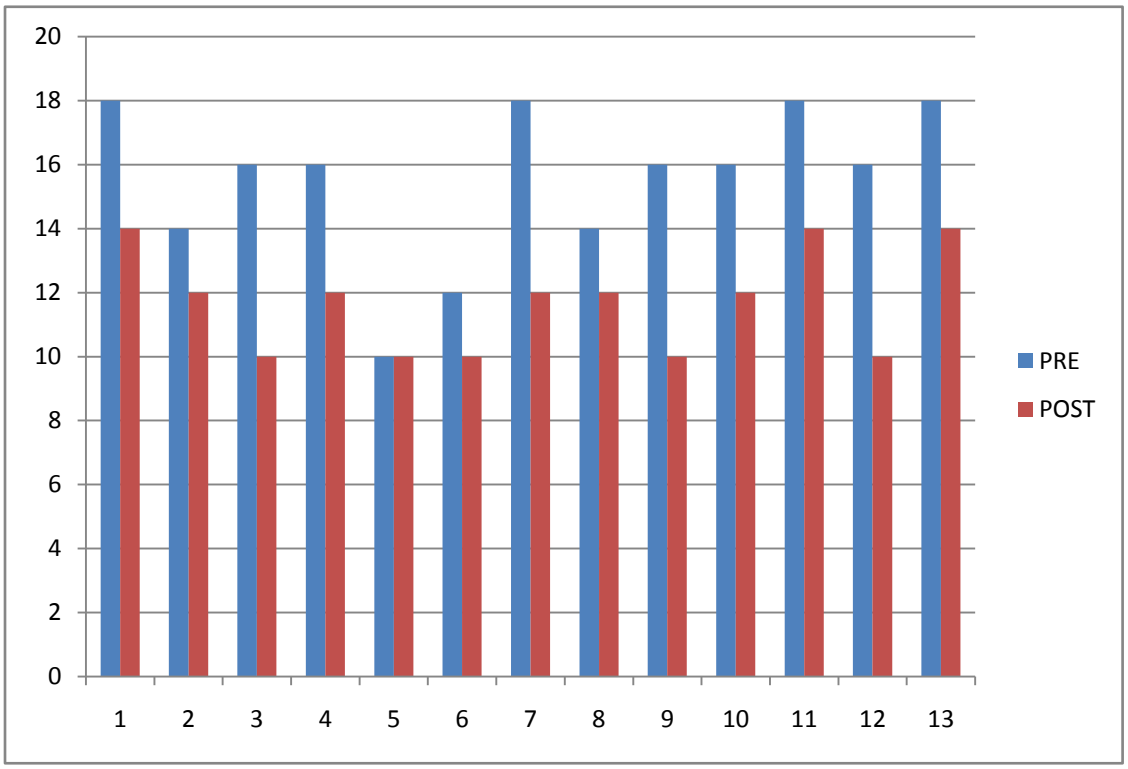
TABLE - 6

S.NO	VALUES	LEFT EYE (IOP) (MM OF HG)	
		PRE EXERCISE	POST EXERCISE
1	MINIMUM	10	10
2	MAXIMUM	18	14
3	MEAN	15.54	11.69
4	STANDARD DEVIATION	2.47	1.60

LEFT EYE

HISTOGRAM SHOWING PRE AND POST EXERCISE INTRAOCULAR PRESSURE CHANGES IN FEMALES:

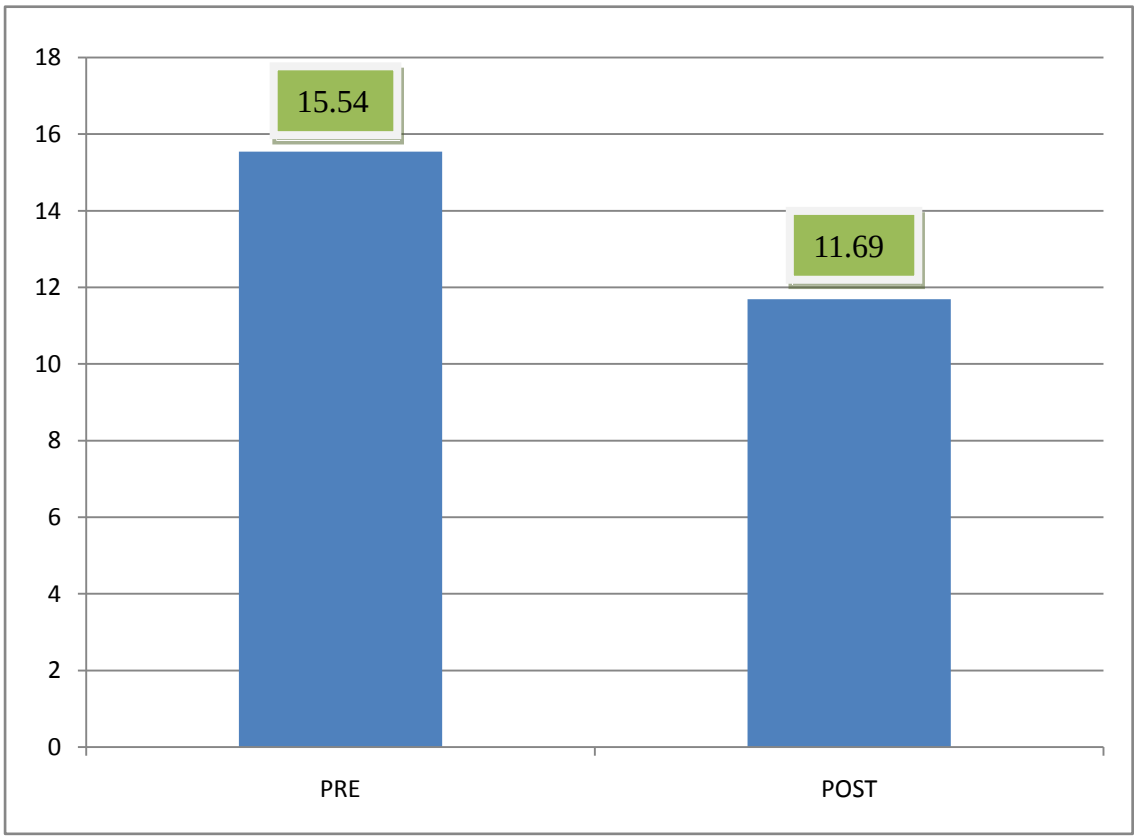
Histogram 11:



LEFT EYE

HISTOGRAM COMPARING THE MEAN VALUE OF PRE AND POST EXERCISE INTRAOCULAR PRESSURE IN FEMALES:

Histogram 12:



RESULT ANALYSIS:

Histogram 1: shows the comparison between the pre exercise and post exercise IOP of right eye in both males and females. It shows a significant decrease in IOP after exercise in most of the individuals.

Histogram 2: shows the mean value of IOP of right eye in both males and females before and after exercise. The difference between the mean value noted before and after exercise is around 3.3 mm Hg.

Histogram 3: shows the comparison between the pre exercise and post exercise IOP of left eye in both males and females. It shows a significant decrease in IOP after exercise in most of the individuals.

Histogram 4: shows the mean value of IOP of left eye in both males and females before and after exercise. The difference between the mean value noted before and after exercise is around 3.48 mm Hg.

Histogram 5: shows the comparison between the pre exercise and post exercise IOP of right eye in males. It shows a significant decrease in IOP after exercise in most of the individuals.

Histogram 6: shows the mean value of IOP of right eye in males before and after exercise. The difference between the mean value noted before and after exercise is around 3.21 mm Hg.

Histogram 7: shows the comparison between the pre exercise and post exercise IOP of left eye in males. It shows a significant decrease in IOP after exercise in most of the individuals.

Histogram 8: shows the mean value of IOP of left eye in males before and after exercise. The difference between the mean value noted before and after exercise is around 3.37 mm Hg.

Histogram 9: shows the comparison between the pre exercise and post exercise IOP of right eye in females. It shows a significant decrease in IOP after exercise in most of the individuals.

Histogram 10: shows the mean value of IOP of right eye in females before and after exercise. The difference between the mean value noted before and after exercise is around 3.54 mm Hg.

Histogram 11: shows the comparison between the pre exercise and post exercise IOP of left eye in females. It shows a significant decrease in IOP after exercise in most of the individuals.

Histogram 12: shows the mean value of IOP of left eye in females before and after exercise. The difference between the mean value noted before and after exercise is around 3.85 mm Hg.

DISCUSSION:

In our study we noticed significant reduction in intraocular pressure measured immediately after an isotonic exercise (walking in treadmill for 30 minutes) in all students.

Ashkenazi et al who studied the effect of a continuous 110-km march with a 20-kg backpack(isotonic exercise) load on intraocular pressure (IOP), plasma osmolarity, blood lactate, pH and observed significant decrease in intraocular pressure.

In 2004 Brownlee P et al analysed the changes in IOP and ocular blood flow during exercise. They classified the subjects based on the level of exercise (mild, moderate, severe) and age group. They noticed significant reduction in IOP in all age groups.

Lipkova J et al in 2008 done a study in 15 open angle glaucoma patients(8 in target group and 7 in contol group). Patients in target group undergone regular exercise (3 times a week) for 8 weeks. They noticed that exercise doesn't have any influence on IOP. IOP was stable during the study periods.

Mohammed Ehtesham et al compared the decrease in IOP between individuals with BMI greater than 22 and individuals with BMI less than 22 by doing exercise. They noticed that there was a significant decrease in IOP by doing exercise in individuals with BMI greater than 22.

Similar to our study, Sunitha et al analysed the exercise induced IOP changes in 35 males without any ocular or systemic abnormality in the age group of 18-25 years. They measured IOP before and immediately after exercise and at 5 minutes and 10 minutes after exercise. There was a statistically significant decrease in IOP immediately and at 5 minutes after exercise.

In 1970 Marcus et al done a study to find out the factors responsible for decrease in IOP during exercise. They included 12 volunteers (6 males and 6 females) who were subjected to isotonic exercise and sodium lactate infusion on separate occasions. Apart from decrease in IOP they observed increase in serum lactate levels, increase in serum osmolarity and fall in blood pH on both occasions.

A study conducted by Natsis k et al states that there is a statistically significant reduction in IOP during jogging regardless of the usage of b-blocker or a prostaglandin analogue or an α-agonist in both

normotensives and glaucoma patients.

In 2002 Vieria GM et al did a study effect of exercise on IOP in 25 healthy volunteers without glaucoma. They noticed a small but significant reduction in intraocular pressure.

In june 2003 Price et al noticed a significant reduction in intraocular pressure after treadmill exercise which returned to preexercise levels after 30 minutes of exercise.

Risner D (2009) et al stated that dynamic exercises causes acute reduction in intraocular pressure which returns to pretrained levels after 1 month of cessation of exercise.

A study conducted by SA Read et al on 20 individuals (10 myopes and 10 emmetropes). All were subjected to moderate intensity , low impact dynamic exercise. They noticed significant reduction in IOP, axial length and ocular pulse amplitude but there was no difference in the changes between myopes and emmetropes.

Gungor K et al stated that due to polymorphic nature of beta 2 receptors IOP response to exercise differs depending upon the type of receptors stimulated.

Qureshi IA in 1996 stated that the acute initial decrease in intraocular pressure depends on the intensity of the exercise and not on the duration, quantity of the exercise, blood pressures or body mass index.

Williams PT did a prospective epidemiologic cohort study in 29,854 male runners without glaucoma in the mean age group of 45 years. They done a study for a period of 7.7 years and the IOP decrease is directly proportional to the intensity and duration of exercise. Runners who ran 3.6 to 4 m/s and 4.1 to 4.5 m/s had 29% and 54% decreased risk of developing glaucoma.[94]

SUMMARY:

With the aim of evaluating the effect of exercise on the intraocular pressure, we started this study with the study group comprising 46 students which included 33 males and 13 females. After getting the institutional ethics committee approval and due consent from the students the methodology was framed out. The methodology included the application of the anaesthetic (Aurocaine) eye drops and fluorescein on both eyes followed by which the intraocular pressure was measured by goldmann applanation tonometer before and after exercising in the treadmill for about 30 minutes with the speed of 7 km/hour. The observation and results of the study were that the mean intraocular pressure decrease in right eye of the males and females, left eye of males and females were 3.21 and 3.54 mm of Hg, 3.33 and 3.85 mm of Hg respectively, the total (males and females) fall in mean IOP of right eye and left eye was 3.30 and 3.48 mm of Hg respectively. This decrease was found to be statistically significant($p<0.05$).

CONCLUSION:

Thus from the above study we are concluding that there is a significant decrease in intraocular pressure after exercising for about 30 minutes in treadmill with the speed of 7 km/hr. Glaucoma is the second leading cause for blindness in the world and is also considered as the silent thief of sight. So by this study we stress the importance of exercise in controlling the glaucoma with the future aspect of adding the exercise as an adjunct to antiglaucoma drugs in the treatment of glaucoma.

LIMITATIONS:

Sample size was limited due to time constraint. The samples should have been included the glaucoma patients. The intraocular pressure was not measured in a serial interval after the acute fall due to exercise to assess the return of intraocular pressure to the normal baseline. Additional parameters such as axial length of the eye ball, pulsatile ocular blood flow, serum osmolarity and blood pH were not measured due to time constraint. Genetic analysis was not done.

RECOMMENDATIONS:

Sample size should be increased. Glaucoma patients should be included in the future studies. Intraocular pressure should be measured in a serial interval after exercise till it reaches the normal baseline. Trials should be made by comparing the exercise and antiglaucoma drugs in reducing the intraocular pressure and exercise as an adjunct to antiglaucoma drugs in reducing the intraocular pressure. Genetic analysis should be done. Multicentric studies in and around the tamilnadu will increase the credibility to this study.

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MASTER CHART

A) **MALES**

S.No	NAME	REG.No	IOP BEFORE EXERCISE		IOP AFTER EXERCISE	
			RE	LE	RE	LE
1	SATHEESH	11BN005	14	12	12	10
2	SHAM PUNKATZ	11BN066	12	14	12	12
3	SATHISH	11BN055	16	14	10	10
4	NITHYA BALA	11BN042	10	12	10	10
5	JIM ROLAND	10BM109	16	16	12	12
6	PRADEEP	10BM080	14	14	10	10
7	KANMINI ANAND	10BM155	10	10	10	08
8	AVINASH PRAVIN	10BM136	16	16	10	12
9	RAGHURAM VIKRAM	10BM140	10	10	10	10
10	PRABHAKARAN	10BM081	18	18	12	12
11	HARIPRAKASH	10BM093	08	10	08	08
12	MURALI	10BM066	16	18	14	14
13	ABILASH	10BM006	14	14	12	10
14	VIGNESHWARAN	10BM118	18	16	12	12
15	VENU	10BM016	14	14	10	10
16	UNNIKRISHNAN	10BM141	12	12	10	08
17	VISHNU	10BM004	14	12	10	10
18	SUBRAMANIAN	10BM034	18	18	14	14
19	SHYAM SUNDAR	10BM149	16	16	12	12
20	SABARIRAJ	09BMO53	14	14	10	10
21	SAMPATHKUMAR APPUCHAMI	10BM101	12	10	08	08
22	RUPAK	10BM057	16	18	12	12
23	NAVNEETH	10BM055	14	16	10	12
24	HEMANTH KUMAR	10BM067	14	14	10	10

25	GOKUL	10BM104	12	12	08	10
26	MANIKANDA PRABHU	10BM107	14	16	12	12
27	ILANCHEZHIYAN	10BM091	18	16	10	10
28	INBASAGARAN	10BM047	14	12	12	12
29	PRASHANTH	10BM045	12	14	10	10
30	VIGNESH	10BM033	12	10	10	08
31	VIVEK KOUSHIK	09BM125	18	18	16	14
32	VIJAY BALAJI	09BM117	16	14	12	10
33	VIGNESH	09BM126	14	14	10	12

B) FEMALES

S.No	NAME	REG.No	IOP BEFORE EXERCISE		IOP AFTER EXERCISE	
			RE	LE	RE	LE
1	YUVASREE	09BM001	18	18	14	14
2	TAMILMOZHI	09BM107	16	14	12	12
3	THANARASI	09BM043	16	16	10	10
4	VEDHASANKARI	09BM019	14	16	12	12
5	SWETHA RAVICHANDRAN	09BM022	10	10	08	10
6	SAKTHISREE	09BM191	14	12	10	10
7	VARSHINI	09BM031	18	18	16	12
8	SAI SATHYA	09BMO91	16	14	14	12
9	SHANTHINI	09BM159	16	16	10	10
10	SWATHI	09BM033	14	16	10	12
11	SURYAPRABHA	09BM122	16	18	14	14
12	VISHNUPRIYA	09BM118	14	16	10	10
13	SWATHE ANAND	09BM164	16	18	12	14